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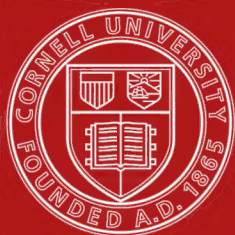
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TEXT BOOK
OF
VETERINARY MEDICINE

BY
JAMES LAW, F.R.C.V.S.

Director of the New York State Veterinary College,
Cornell University, Ithaca, N. Y., Emeritus.

VOL. I.

THIRD EDITION

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PREFACE TO THE SECOND EDITION.

THIS work has been so favorably received in all parts of the English-speaking world, that it is with a special satisfaction that the author responds to the demand for a second edition. In the new edition he avails of the opportunity to carefully revise the first, so as to bring it in line with the latest advances in veterinary medicine. To accomplish this over one hundred and fifty pages have been added to the first volume, embracing the more important achievements made since the first edition was published, and the author bespeaks for the new issue, a continuance of that appreciation which has been so liberally accorded to the first.

J. L.

New York State Veterinary College,
Cornell University,
Feb. 1, 1905.

PREFACE TO THE THIRD EDITION.

IT is with profound satisfaction that the author responds to the demand for a third edition, as the rapid advance of Veterinary Medicine necessitates constant revision to keep a work of this kind up to date. To meet this need the whole of Vol. I has been carefully revised, and the succeeding volumes will be dealt with in a similar way so as to justify the growing appreciation which the veterinary profession is showing to the work.

J. L.

Cornell University,
Ithaca, N. Y.
July 9, 1910.

VETERINARY MEDICINE.

OBJECTS AND METHODS OF STUDY.

Pathology—general—special. Morbid anatomy. Pathological chemistry ; Disease. Health. Death—Somatic—partial—necrosis. Syncope. Apnoea. Asphyxia. Coma. Death from old age.

The principles and practice of Veterinary Medicine should embrace all that is known of the causes, nature, symptoms, prevention and cure of disease in domestic animals. Incidentally it includes diagnosis and prognosis.

Pathology is the science which tells of the causes and nature of disease, and the functional and structural changes by which it is characterized. In modern usage the term pathology is understood to refer to the intimate nature of disease, but this necessarily involves an enquiry into its sources and the predispositions to its occurrence ; its phenomena whether in changes of function or structure ; and its results in the form of perverted function, structural changes, degenerations, dependent disorder, etc. The field of pathology is further divided into general pathology and special pathology.

General Pathology treats of disease processes in their generic form, and as they appear in many different diseases. Thus inflammation and fever are the prominent phenomena in a great many different diseases which differ in their seats, their causes, manifestations and results. Inflammation and fever are therefore subjects of general pathology. Similarly all forms of degeneration—fatty, fibrous, calcic, amyloid, etc., are disease processes found in many different organs and under very varied conditions and they are accordingly included in general pathology. Hypertrophy and atrophy are also possible in every organic tissue irrespective of kind or seat, they belong therefore to this particular field.

Special Pathology on the contrary is confined to a particular disease and not only elucidates the causes, phenomena and results of such disease, but seeks to do this in such a way as to differ-

entiate this malady from all others however closely related to it. Thus inflammation of a bone is known under the general name of osteitis, this may be due to a great variety of different causes, and each would have its own special pathology. The osteitis of simple mechanical injury is essentially different from the osteitis of rheumatism, of purulent infection, of tuberculosis, of actinomycosis. So with the inflammations of every other tissue. Each may suffer from a variety of inflammations, springing from different causes, attended with characteristically unlike tissue changes and tending to different tissues, and every one of these forms has therefore its own special pathology.

General Pathology may be said to deal with typical disease processes to a large extent irrespective of the individual disease in which they may appear, while **Special Pathology** deals with the morbid phenomena which distinguish the individual malady from all other diseases however closely allied to it.

Pathological (Morbid) Anatomy deals with structural changes, the cause, the accompaniment or result of disease. These morbid changes are microscopic or macroscopic. Both constitute morbid anatomy, but the microscopic alterations come under the special name of morbid (pathological) histology.

Pathological Chemistry is that branch of pathology which treats of chemical changes produced by disease in the blood, lymph, tissues, secretions and excretions. It demands a previous knowledge of the condition of these tissues and fluids in health, in the particular genus of animal and under the same dietary and environment. Physiological Chemistry is therefore an essential prerequisite to pathological chemistry, just as anatomy, physiology and histology are indispensable to the appreciation of pathology and morbid anatomy.

Disease is an injurious deviation from the normal function or structure. The morbid process resulting in disease is usually in the nature of a modification of the normal or physiological condition, so that it is often difficult to set the exact limits of health and disease. What is a purely physiological process under given conditions, would be distinctly pathological under others. The free kidney secretion of cold weather and the profuse perspiration of a hot season are both purely physiological and in the main balance each other. Each under its special environment fulfills

an essential work in eliminating from the system toxic materials which would prove hurtful if retained, and thus each is not only physiological but beneficial. If, however, they occurred, not in this mutually compensatory manner, but simultaneously in this profusion, they might well be dreaded as morbid conditions. Again if either were to occur apart from its normal causative environment, if for example the polyuria appeared in hot weather and the perspiration in cold, the phenomenon might fairly be called pathological. In any case if the excessive secretion induced a lowering of the general tone of health the process would be essentially a morbid one. In pronouncing therefore upon a morbid process, one must take fully into account the corresponding physiological process, the attendant conditions, and whether the result is injurious or otherwise.

The same is true of structural changes. What under given conditions would be essentially a morbid structure, might under other conditions be a simple adaptation to an unwonted environment, and a means of protection from injuries that would otherwise accrue. Excessive growth of cuticular tissue in the epithelioma, wart or corn is injurious and essentially pathological, while the callus on the camel's knee or the workman's palm is purely protective and physiological. The local development of a mass of fatty tissue in the average man or beast is a disease, but the tendency to the uniform deposition of fat in the connective tissue of the improved breeds of meat producing animals, is the happy culmination of a long continued and skillful selection and regimen, without which the livestock industry of today would be a grievous failure. To constitute disease, therefore, modified function must be not simply a compensating increase, decrease, or other change, and it must be in some way injurious to the animal economy. Similarly, to constitute disease, modified structure must be other than a simple protective or beneficial change, it must not be a simple evolution in the nature of accommodation to the environment but it must be a cause of injury to function or a distinct deformity.

Health may be said to be the harmonious exercise and mutual balance of all the bodily functions, and any interference with such mutual exercise or balance may be said to constitute disease. But as health passes into disease by insensible gradations,

there is of necessity an extensive borderland which often cannot be allotted to one condition or the other, but which must often be left a disputed territory.

Again certain animal constitutions are innately strong and robust, while others are weak and feeble, yet the delicacy of the latter cannot be set down as actual disease, and by maintaining a due balance between the functions, a fair measure of health and even long life may be secured.

Death as the result of disease may be either *partial* or *somatic*.

Partial or local death may be *molecular* as in **ulceration**, or it may effect an organ or part of an organ, as in **necrosis**, **sphacelus**, or **sloughing**. **Somatic death** is a loss of vitality of the entire body and is manifested by a complete cessation of the bodily functions, including that of nutrition. Usually the arrest begins with one of the great vital processes, in advance of the others, and thus in different cases, we have *death beginning at the heart, at the lungs, and at the brain*.

Death from syncope or fainting, begins at the heart, which loses its irritability or contractility, or is seized with a tonic spasm. If there has been lack of contractility, the heart is found after death in a flabby, soft condition, and quite frequently filled with blood. If heart-spasm, it is contracted, firm, and empty or nearly so. Syncope may result from severe nervous shock (emotional), from the electric current, from insolation, or from heart sedatives like chloroform, or nicotine. It may, however be but the culmination of a gradually advancing debility, from exhausting diseases, from fatty degeneration of the cardiac muscles, or from starvation, or anæmia. Again the exhaustion coming from profuse hæmorrhage, or from violent over-exertion is a cause of fatal syncope.

In **death beginning at the lungs** (apnoea, asphyxia, or suffocation), the blood failing to receive oxygen and to give up its carbon dioxide is unable to maintain the various functions of the body and the arrest of the other vital processes speedily follows. The arrest of the respiratory process may occur from nervous shock, but more commonly it results from choking, strangulation, drowning, or the action of irrespirable gases. In diseases of the heart and lungs it is liable to occur from the obstruction of the pulmonary circulation and from the depression of the respira-

tory nervous centres. After death the lungs are found gorged with dark red—almost black—blood, which likewise distends the right heart and systemic veins, and all mucous and serous membranes have a dark red, congested aspect. When breathing has been arrested by mechanical violence there are, first, active contractions of the respiratory muscles, but no loss of consciousness; then as the brain becomes charged with venous blood, consciousness and volition are lost and convulsive movements ensue. Later still there is no respiratory effort nor convulsions, but the heart continues to beat for two or three minutes longer.

In death beginning at the brain (Coma) the sensory functions fail first, as evidenced by drowsiness, stupor, or complete insensibility, while the movements of heart and lungs are still temporarily continued. Pressure on the brain by a fractured bone or blood clot, or in cases of violent congestion or the rapid growth of tumors, usually operates in this way. It may also result from the direct action of certain poisons, like opium, belladonna, or chloroform, or the ptomaines or toxins of bacteria. Causes acting on the brain may, however, lead to death by syncope or asphyxia when the nerve centers presiding over circulation or respiration are the first to feel the full effects of the pressure or poison.

Death from old age, with a gradual failure of the natural processes of nutrition and tissue-growth, and the occurrence of atrophy and various degeneration of the organs is not a common occurrence in domesticated animals, so that it may be dismissed without further notice.

Actual somatic death is marked by the cessation of breathing and pulse, the dilated pupils and semi-closed eyelids, the coldness and palor of the visible mucous membranes and skin, and the clenching of the jaws with slight protrusion of the tongue. Yet these symptoms may be present in syncope and it may even be impossible to detect the beats of the heart, though the subject still lives. Pressure of the finger on a white portion of the skin, or on a mucous membrane may give a further indication. If the indentation made by the finger is slowly effaced and if the blood again slightly reddens the part the presumption is against death. Even this is not infallible, since by pressure of gas in the internal cavities or deeper blood vessels, the blood may be forced back

into the surface capillaries giving an appearance of circulation, after actual death. On the other hand any exudation or œdema will retain the imprint of the finger even in life. The general relaxation of the muscles and their lack of response to electric stimulation, and the setting in of cadaveric rigidity, and later still of putrefaction give more conclusive evidence of dissolution.

ETIOLOGY : CAUSES OF DISEASE.

Causes—simple—complicated : Proximate ; Remote : Predisposing—race, genus, family, heredity, individual, environment, food, age, sex, temperament, idiosyncrasy, debility, plethora, interdependence of organs, embolism, mechanical influence. Exciting causes, intrinsic, extrinsic, inherent, acquired, heredity, dentition, heat, cold, atmospheric conditions, electricity, moisture, dryness, dust, darkness, light, soil, food, water, inaction, over-exertion, mechanical causes, poisons—mineral—vegetable—animal, microbes, contagious, infectious, epizootic, enzootic, sporadic, panzootic, zymotic, mediate contagion, bacterial poisons.

The causes of disease are simple or complicated; and in the latter case a single factor may be altogether harmless unless associated with another which also may have been innocuous alone. For example : the infecting germ of glanders (*Bacillus Mallei*) is harmless to the ox which lacks the predisposition to the disease :—feeding buckwheat is harmless to the dark-skinned animal, but is injurious to the white skinned, if exposed to sunshine :—the chicken can bear with impunity exposure to cold or to the bacillus anthracis, but it cannot endure these two etiological factors combined. It follows that one cannot predict the same result from the same cause in every case. Yet with all concurrent conditions the same the result will follow with mathematical certainty. This will serve to illustrate the value of thoroughness in etiological knowledge, as the basis of a sound pathology.

Etiology is primarily divided into proximate and remote. Remote causes are again divided into perdisposing and exciting.

Predisposing Causes are such as induce a condition of the system or of a particular organ or group of organs which renders them specially susceptible to a disease. This may be characteristic of the *race* or *genus* of animal, thus the genus *bovis* alone suffers from lung plague, the genus *equus* from bursatti, and ruminants from Rinderpest. It may be a *family trait*, (*hereditary*) hence we see certain families of both men and cattle cut off by tuberculosis, while other adjacent ones largely escape. It may be an *individual peculiarity*, thus some subjects have a congenital insusceptibility to a given disease, from which others of the same family suffer, and one who has passed through a self-limiting disease like measles, cowpox or anthrax is rarely attacked a

second time. Again predisposition may be due to *environment* as when we find herds in damp and exposed localities obnoxious to rheumatism, and horses in dark mines exposed to specific ophthalmia. It may be the result of *food* as when the flesh-fed fox or rat resists anthrax and the farina-fed one falls a ready victim. *Age* may predispose, early youth being remarkably susceptible to parasitism and bacteridian infection, and old age to fractures and degenerations. *Sex* is inevitably a cause of limitation of disease as the females and males can only suffer from the disease of their respective sexual organs. Again of diseases common to both sexes certain nervous and digestive disorders are common in connection with gestation, and certain calculous diseases in connection with the long and narrow urethra of the male. *Temperament* has a marked influence, thus the sanguine or nervous race-horse or hound shows a marked predisposition to diseases of the heart, lungs and brain, and to a sthenic type of inflammation and fever, while the heavy lymphatic draught-horse has a proclivity to diseases of the lymphatics and skin. *Idiosyncrasy* is closely allied to temperament, but the condition may be less manifest, and the peculiarity is only recognized by the results, as when a man is poisoned by sound fish or raspberries. *Debility* whether from deficiency or poor quality of food, on the one hand, or from overwork, filth, dampness or disease on the other must be looked upon as strongly predisposing to certain diseases, such as tuberculosis and glanders. *Plethora* which charges the blood and tissues in a different way with effete organic products, lays the system especially open to certain diseases like black quarter in young cattle, and parturition fever in cows. *Disease of one organ* often predisposes another organ through interdependence of function, as when torpid or congested liver leads to portal and intestinal congestion, diseased teeth to digestive disorder, imperfect hæmotosis to kidney trouble ; in other cases blood clots or bacteria from one pathological center may be arrested in the blood vessels of a distant organ and start new foci of disease (embolism, metastasis) ; in still other cases the impairment of the healthy function in one organ acts injuriously on another, as when emphysema or other disease of the lungs forces the blood back upon the heart causing dilatation with atrophy of the walls. *Previous disease in a tissue* leaves for a time an impairment of

structure and function which may become the essential predisposing cause of the effective operation of a morbid factor. *Mechanical* action on a part may predispose to disease, as for example, by reducing its circulation and nutrition and thereby directly impairing its power of resistance to other inimical agencies. Not infrequently a pus microbe lies deep in the cuticle or even in the tissues without harm, until there occurs a bruise, or a bony fracture when it at once develops a focus or purulent infection (abcess).

Exciting Causes are the immediate occasion of particular diseases. Like the predisposing causes they may be *intrinsic* or *extrinsic*, and the first may be **inherent** or **acquired**.

Among **inherent** causes are certain of those already named as predisposing causes, but which have come to be forcible enough to develop disease without the intervention of any other observable factor. Thus a *hereditary* monstrosity (redundancy or defect) will appear in successive generations without any apparent additional cause. The appearance of white calves in herds of black cattle, after the whitewashing of their stables shows a similar hereditary operation though the result is not in this case pathological. The birth of blind foals from blind sires or dams, or of foals with distorted feet from mares suffering from severe chronic foot lameness are true pathological sequences, in which the exciting cause is congenital and operates during intrauterine life. *Dentition*, as an attendant on early life is often a directly exciting cause, from direct injury by entangled or retained teeth that should have been shed, by fever aroused by the active local changes, or imperfect mastication or insalivation leading to consequent indigestion; in puppies and kittens convulsions are not uncommon as a result.

Extrinsic Causes are such as operate through the environment. *Heat*, if excessive and prolonged, relaxes and exhausts the system and exerts a direct influence on the process of sanguification so that it may become the direct cause of a variety of diseases. As the result of extensive burns, dangerous congestions of internal organs are liable to occur, and even the prolonged heat of summer often superinduces hepatic and gastric disorder, diarrhoea and dysentery. Fat cattle in uncovered cars or yards under a hot sun and with no breeze suffer extensively from in-

solution, the temperature of their bodies rising to 110° to 112° Fah. and even higher. *Cold* is equally potent. With a temperature below zero Fah., the iron bit will freeze the buccal mucous membrane, and cause extensive erosion of the mouth. The cold of salted snow or ice will freeze the feet causing sloughing of the skin above and around the coronet and shedding of the hoofs, and predisposing to fatal septic infection of the wounds. On the system at large, cold causes retrocession of the blood upon the internal organs, and endangers the occurrence of acute disease in any structure which is already debilitated or otherwise susceptible. The nervous effect of the chilling of the skin is often the unbalancing factor which sways the scale in favor of disease, which the system was able to resist until this disturbing element was introduced. The sudden chill from passing out of the warm barn into the frosty air, from plunging into icy water, from standing in cold rain or sleet, from standing in a draught of cold air, especially when perspiring, is a fruitful source of many diseases. In the cow, lying with the udder on a cold stone may be the starting point of mammitis. The effect of sudden chill is well exemplified in the great prevalence of diseases of the respiratory organs at the change of the seasons in spring and autumn when the vicissitudes of temperature are greatest, and the system unprepared by habit to bear the sudden change. Even contagious diseases may often claim a chill as a controlling factor. The microbes, already present, though harmless in a strongly resistant system, become at once pathogenic when circulation, nutrition and nervous control are impaired by exposure to cold. Again it must be noted that exposure to cold has a tendency to cause disintegration and solution of the red blood globules, and that certain animals are especially susceptible to this influence. *The condition of the atmosphere* is often a direct cause of disease as when charged with offensive or irritating gases, the result of decomposition of organic matter, with sewer or cesspool emanations, with deleterious gases from chemical works, telluric sources, or fires. A low state of health, a local irritation in some part of the air passages, or even a speedy asphyxia may be the outcome of such atmospheric conditions. Again the presence of solid particles of a more or less irritant, septic or infecting kind, proves the starting point of various diseases. The stone cutters'

phthisis, and the sand granule ophthalmia are familiar examples of the irritant, which often acts through the dust of the highway. Of the infecting particles we have the germs of cattle lung plague, of infecting ophthalmias, and of tuberculosis carried with the dry dust and inhaled. Of toxic agents borne on the atmosphere we see the compounds of arsenic, mercury and lead. Moisture and dryness of the air induce respectively a lymphatic constitution and low tone of health, and a nervous constitution and a tendency to neurosis, ophthalmias, and skin diseases. The pressure of the atmosphere has a profound effect on animal health as seen in the extreme troubles of the heart and circulation in the diving bell, and the respiratory, hæmorrhagic and brain affections of high altitudes. A low barometer is attended by nervous disorders (neuralgia) (S. Weir Mitchell). Surgical operations do best with a high or rising barometer (Adinell, Hewson). The electrical tension of the atmosphere shows familiarly, in man, in the feeling of heaviness, dullness and malaise that precedes the bursting of a thunderstorm and the relief that follows its termination. To this influence many of the domestic animals are incomparably more sensitive than man, as witnessed in the disposition of swine to hide in their pens or under litter on the approach of the storm, the nervous disturbance even to abortion of certain ewes which are heavy in lamb, and the great discomfort and even piteous cries of some domestic felines on such occasions. The greatest electric tension is seen in the drier climates, where the air, robbed of its moisture, proves a poor conductor and equalizer, and the positive and negative electricity get stored up separately in air, cloud and soil. The presence of ozone in the air, as a habitual concomitant of electric discharges, has been supposed to be a disturbing influence, since it is distinctly irritating to the mucous membrane when present in excess, but such excess apart from its artificial production is highly improbable. As habitually met with it is antiseptic and health giving. *Darkness* always deteriorates the general health, producing bloodlessness and pallor. *Light* is usually invigorating, yet bright sunshine falling upon the eyes from a window in front of the stall, or in the open air when the face is turned up by an overdraw check-rein, or reflected from white dust and, above all, from snow, will often induce inflammation and blindness.

Soils are often potent etiological factors. Dense, damp, cold, undrained soils, are habitually covered by a stratum of cold air, saturated with moisture, which greatly lowers the vital stamina. Damp clays, and waterlogged soils of various kinds, rich in organic debris, are the natural homes of various pathogenic microbes, such as those of ague, anthrax, milk sickness, actinomycosis and yellow fever. Well drained sandy or gravelly soils are usually healthy, unless they contain a great excess of decomposing organic matter. Again, soils with an excess of alkaline or other mineral matter may prove deleterious, and those on the magnesian limestone often harbor the poison of goitre, and cretinism, and favor the occurrence of urinary calculus. *Faulty food and feeding* in the domestic animals are chargeable with many diseases. Stock often fall off in condition, in the hands of one feeder, when the same food given with regularity and judgment by a more careful feeder would keep them in the highest health. Hay and grain which are musty and filled with cryptogams and their products, are common causes of disorder of the stomach, the kidneys, the nervous system or of general nutrition. Smut and ergot at certain stages of their growth or grown under given conditions cause nervous disorders, abortion, and gangrene of the extremities. A long list of vegetable poisons may mix with fodders, and animal poisons with the food of the Carnivora. A number of standard fodders may be poisonous at certain stages of growth, as partially ripened perennial rye grass, millet, Hungarian grass, vetches, etc. *Water and deprivation of water* are fertile causes of illness. Ruminants cannot chew the cud when deprived of water, hence impaction of the first and even of the third stomach with fermentations, tympany and other disorders. Horses suffer more from a full drink of water after a feed of grain, the unchanged albuminoids being carried on into the intestines, and both gastric and intestinal indigestion induced. Sheep suffer fatal fermentations after drinking the alkaline water of the Plains; cattle have diarrhoea and dysentery from selenitious, or from stagnant and putrid water; and the water from the dolomite is the usual channel of the goitre poison. Certain germs like the plasmodia of malaria, and comma bacillus have their natural home in impure water, or in the insects that breed there, and others like anthrax bacillus survive in the mud and

silt at the bottom of wells, ponds, and rivers and enter the system in the water. *Compulsory rest* in a stall often induces torpor of liver and bowels, general muscular debility, and fatty degeneration especially of the liver and heart. A few months of the swill feed, hot atmosphere and absolute rest in a distillery stable usually ruin cattle for stock purposes. *Over exertion* on the other hand is prolific of illness. Exhaustion of the muscles, congestion, inflammation, cramps, congested lungs, heart failure or rupture, apoplexies and other hæmorrhages are among the resultant maladies. Auto-poisoning is another result shown in equine hæmoglobinæmia, and the fever of leucomaines. The excessive development of sarcolactic acid from muscular work may render an insusceptible animal susceptible to the anthrax bacillus. *Mechanical causes* would include overexertion, in the production of strains, fractures, and other injuries. They would also include impaction by foreign bodies, calculi, and ingesta, friction of folds of skin or by harness and other objects, and pressure which leads to absorption and atrophy. To these must be added *poisons of vegetable, mineral and animal origin* and the *microorganisms* which act as injurious ferments within the animal body. These will be treated more fully later on. Of the microorganisms it may be said here, that they are almost certainly the cause of all transmissible diseases. These diseases are variously named on the basis of different ideas. They are **enthetic**, that is implanted as a seed is planted in the ground to grow and multiply. They are **zymotic** or fermentative because the essential cause multiplies and is propagated like a ferment. They are **contagious** because propagated by contact mediate or immediate. They are **infectious** when transmitted, not alone by contact but through the atmosphere. They are **epizootic** because they tend to attack animals generally or a given genus or family of animals generally when these are exposed to the infection. They are **enzootic** when confined to the animals in a given locality, the soil or conditions of which are favorable to the preservation of the germ in pathogenic potency, or to the production of a special susceptibility in the animal system. They are **sporadic** when each case occurs without any causal relation to another. They are called **panzootic** when they attack all animals without apparent preference. The term *panzootic* is also used to describe those recrudescences of a disease or cycles of

exalted pathogenesis which are observed in contagious diseases, which frequently last for years and again give place to a period of benignancy. Such cycles, of malignancy and benignancy, may be due to modified environment acting either on the disease-germ or the animal system, or on both simultaneously.

The terms *enthetic*, *zymotic*, and *contagious* best express modern views of the nature of these maladies. The term *infectious* when used to express a gaseous or otherwise intangible (unorganized) body, or influence transmitted through the air, necessarily excludes the particulate, living, self-propagating germ upon which the transmissibility of the disease depends. A chemical, electrical, or other body or influence generated outside the animal body, cannot well be conceived of as reproducing itself within the animal body, but must act like any other ectogenous poison, according to the size of the dose and the frequency of its exhibition. This might create an *enzootic* disease, but would lack all the qualities of a contagious affection since it could not spread from a victim when taken elsewhere and turned among animals which would prove equally susceptible if placed within the infecting area. Suppose on the other hand we apply the term *infectious* to diseases in which the levity of the particulate living germ allows of its being inhaled into the body of the susceptible animal, the case becomes one of simple *mediate contagion*, the air acting as the intermediate bearer.

The term *zymotic* conveys a clear idea of the method of increase of the disease germ in the body by the ordinary process of generation. The old doctrine of fermentation by a continuous change, due to contact with dead fermenting matter, as an inflammable body continues to burn by contact with the incandescent portion, has been definitely disproved by the investigations of Pasteur and others, and today we must recognize that every fermentation is the result of the propagation and vital activity of living organisms. This does not ignore the fact that the chemical products or enzymes which are constructed by the vital activity of the microbes, will dissolve or transform organic matter, but in the absence of the microbe no such enzyme can reproduce nor multiply itself and its action must therefore be exactly limited by its amount. The living germ itself is therefore the one effective factor, by which the contagious disease may be maintained and propagated. In its turn the living germ can only come from

a pre-existing living germ. To the scientist of today the doctrine of spontaneous generation is a thing of the past and the aphorism *omne vivum ex ovo* is dominant. The argument drawn from the saccharizing of starch in the germinating seed by the operation of diatase is inapplicable, as the diatase is produced by the living cells of the germinal part of the seed, which are thus the counterpart of the disease germ. No such glycogenic action occurs in the seed that has been boiled or otherwise robbed of its vitality. So with the arguments drawn from the ptyaline of the saliva, the pepsin of the gastric juice, and the trypsin of the pancreatic juice ; each of these is the product of the living cells of the gland by which it is secreted, and cannot increase its own substance in the absence of these cells. Like the enzyme of the bacteridian ferment, these gland products can break down or digest certain organic matters, but in all alike, the only source of the chemical solvent is the living bacterium or gland cell from which the particular product is derived. The toxins of a virulent liquid, after the sterilization of the latter, may still produce most of the lesions and morbid phenomena of the disease, but, although death were to ensue, the body of the victim would not be infecting to other susceptible animals. The parallel between the functions of the secreting animal cells and the disease germs may thus be put in tabular form :

Living Source.	Chemical Product.	Result.
Salivary gland cells	= Ptyaline	= Starch changed to Sugar.
Peptic gland cells	= Pepsin	= Albuminoids changed to peptones in acid solutions.
Pancreatic gland cells	= Trypsin	= Albuminoids changed to peptones in alkaline solutions.
Disease germ	= Toxin : Enzyme	= Morbid phenomena.
Disease germ	=	= Contagious disease.

Further consideration of pathogenic microörganisms will be found in connection with contagious diseases.

Auto-intoxication by poisons produced elsewhere in the system in connection with microbial invasion, or with local disease, functional or structural, and morbid cell-metabolism, may produce new centers of disease of function, or structure, or both.

MEDICAL DIAGNOSIS.

Means of diagnosis. Usual health of the subject. History of the attack. Objective symptoms, interdependent disease, fever, diseases that may be confounded, subsidiary disease, diagnostic signs, organ involved, pathological test injections, course of disease, sporadic or zymotic, result of treatment.

Diagnosis is the determination of the seat and nature of a given disease and its distinction from other morbid conditions. Its importance to the practitioner cannot be overestimated as it occupies the pivotal position between causes, nature, morbid phenomena, and symptoms on the one hand, and prognosis, prevention, and treatment on the other. Unless the conclusions are sound as to causes, nature, lesions, and symptoms, there can be no certain diagnosis, and without a correct diagnosis, prognosis, prevention, and treatment can have no intelligent or scientific basis. The practitioner who finds a dropsical condition and who is satisfied to pronounce it dropsy and institute treatment is abusing his trust. He must find whether this dropsy results from disease of the kidneys, heart, blood-vessels, lymphatics, liver, lungs, bowels, or the structures in which it is shown ; whether it is due to parasites or imperfect sanguification or to other morbid conditions, before he dare prescribe treatment and predict results. So in every other affection ; the failure to make a correct diagnosis opens to the practitioner many doors of error, and he is happy indeed if he can escape the injuring of his patient.

In seeking a sound diagnosis we must attend to the following among other indications :

1st. The habitual state of health of the subject. The genus, breed, age, environment, habits, (pet dog, watch dog, hound, sheep-dog, ox, bull, cow, milch cow, sheep in the field or housed, pig in pen or at large, race horse, draught horse,) diet, regimen, water, work, exposure, etc. as well as the personal equation of temperament, idiosyncrasy, heredity, etc., must all be carefully considered.

2d. The history of the present illness as to its apparent cause, mode of invasion, duration and progress.

3d. The objective symptoms by which it is manifested. All that can be ascertained in the way of symptomatology, local and

general, the probable existence of interdependent disease, and all actual structural lesions and disorders of function should be thoroughly investigated. As supplementary to the more prominent objective symptoms any fever or other constitutional disorder must be sought for; a mental list must be made of the diseases which resemble this one, and these must be excluded one by one by careful attention to the differential symptoms; other diseases which are probably subsidiary to this, should be similarly investigated and excluded; any really diagnostic sign of the suspected disease must be carefully established and the diagnosis finally placed on a solid foundation. The discovery of a constitutional (febrile) disease to which a distinctive name can be given is by no means the end of the diagnosis; the structural lesions of the disease may be largely localized in an unimportant organ where they may remain circumscribed without compromising life, or they may be seated in a vital organ which will render the disease grave to the last degree or necessarily fatal. For example: Anthrax of a dense, dry part of the skin may be a mild local disease; anthrax of an internal organ is usually fatal. Every local complication therefore, should be as carefully diagnosed as the connected constitutional disorder.

But diagnosis cannot always be certain. In the early stages of certain fevers two different forms may be as yet indistinguishable and a day or two may be required to develop differential symptoms. In some occult forms of disease all differential symptoms may fail us. A method of diagnosis which has hitherto been applied only to a few diseases is manifestly capable of much wider application, to diseases attended with a febrile reaction. This consists in a hypodermic injection of a minimum dose of the sterilized and filtered products of the culture of the disease germ, which produces no effect on the healthy system but causes febrile reaction or local inflammation, or both, in the diseased. This will be treated more fully under the respective diseases.

In connection with such a method, but above all when no such resort has been had, the obscure case should be seen frequently, the course, duration, and termination of the disease should be noted; also its tendency—sporadic or epizootic, and finally the result of treatment. This last resort may often secure diagnosis and cure at once, as when a course of iodine cures an obscure actinomycosis.

SYMPTOMATOLOGY. SEMEIOLOGY.

Definition. Symptom. Sign. Constitutional symptoms—local, objective, subjective, direct—idiopathic, indirect—symptomatic, premonitory. Anamnesis. Position. Movements. Decubitus. Acute. Chronic. Fever. Sthenic. Asthenic. State of limbs, muzzle, nose, snout, palmar pad, hoof, bill, digits, mouth. Thermometry. Normal temperature, in doors, in field, at work, in hot season, in nervous subject, in thirst, in youth—age, starvation, plethora, cold, sleep, rest, stimulants, suppressed perspiration, eliminants, antipyretics. Fever temperature, morning, evening, transient elevation, persistent rise, sudden fall—collapse, crisis. Fatal elevation. Rise during defervescence. Pulse. Respiration. Skin, staring coat, pallor, coldness, dryness, harshness, mellowness, pliancy, hide bound, yolk, clapped wool, scurfy, lesions, itchiness, tenderness, loss of hair, emphysema, anasarca, sweat, sebum. Expression, life, dullness, paralysis, dropsy, jaundiced, eye, discolorations, photophobia, amaurosis, pinched face. Nasal mucosa, red, violet, etc., nodules polypi, osseous, disease, pentastoma, œstrus, discharge from teeth—sinuses—actinomycosis—tumors. State of the bowels, kidneys, nervous system.

The usual basis of diagnosis must be a clear and intelligent observation of the symptoms of disease. A *symptom* is an appreciable evidence of disease. A *symptom* however may indicate illness, without affording the means of diagnosis, while the term *sign* is often used for a pathognomonic symptom—one by which the disease can be identified. Used in this sense a *sign* may be said to be a diagnostic symptom.

1. **Constitutional Symptoms** are such as affect the entire system, like a rise of body temperature, or a shivering fit.

2. **Local Symptoms** are confined to a definite area, as redness, tenderness, swelling, ulceration.

3. **Objective Symptoms** include all that can be recognized by the senses of the observer. These alone are available in dealing with the lower animals.

4. **Subjective Symptoms** can only be felt by the patient himself, as pain, giddiness, cold, heat, blindness, numbness. Such symptoms are therefore only fully obtainable from the human patient who can tell how he feels. In the lower animals they can only be matter of inference, thus pain may be inferred from lameness or wincing on pressure, percussion or movement, and giddiness from unsteady gait. The fact that the veterinarian is restricted to objective symptoms renders his task a specially

difficult one, yet this has its compensation, as this very restriction tends to train the observant practitioner to greater skill.

5. **Direct Symptoms (idiopathic)** are those which are connected with the seat of disease, as the redness, exudation, and swelling of inflammation.

6. **Indirect (sympathetic, dependent) Symptoms** are observable at a distance from the actual disease :—as when headache attends on dyspepsia, or lameness in the right shoulder upon disease of the liver.

7. **Premonitory or precursory Symptoms** precede the diagnostic symptoms of some diseases, thus dullness and languor often herald an approaching fever, and the strangles of young horses is often preceded by a general unthrifty appearance, poor appetite and indisposition to exertion.

In observing symptoms as in other things, some have far greater natural ability than others, but in all a careful training will do much to develop and improve the power and habit. A most important thing in such habits is the strict maintenance of a system, not to be followed as a cast iron rule but to be constantly kept in mind and strictly carried out except when sound judgment and experience show it to be unnecessary.

Anamnesis. As a rule the first thing to be learned about a patient is his history, and personal and hereditary characteristics. What are his general health, temperament, previous attacks, hereditary predisposition, environment? Is the site of the building, its condition as regards soil, springs, drainage, structure, ventilation, light, cleanliness such as would favor any particular disorder or class of disorder? Is the patient in high, low, or moderate condition, robust or debilitated, alert and lively or dull and stupid? Have other animals suffered recently, or at a corresponding season, or under similar conditions in apparently the same manner? How long has the patient suffered, were there any premonitory indications of illness, what were the first symptoms, and what symptoms have followed up to the present? Has there been any change of food, water or management that might throw light on the cause? Has there been any change of weather or unwonted exposure to cold, storm, overwork, compulsory abstinence or enforced retention of some secretion? If a female, is she pregnant?

Having exhausted this method, using such lines of inquiry as promise good results in the particular case, the veterinarian is prepared to bring his own powers of observation to bear more directly.

Position and movements will often furnish valuable data. The *horse*, which lies on his ribs, stands obstinately in chest diseases, or whenever there is much interference with breathing. The ruminants and carnivora on the other hand which lie on their smooth or padded sternum, can breathe with comfort in this position and only stand up persistently in the worst cases. The habit of standing day and night is also characteristic of ankylosed back or loins in the soliped. Roached back may be natural, or the result of overwork and slight sprains or injuries of the loins, of ankylosis, of intestinal or renal inflammation, or of certain injuries to the limbs. The extension of the head on the neck may suggest sore throat, chest disease, tumors around the throat, abscess (fistula) of the pole, sprain or spasm of the extensors of the neck, disease of the axoido-atloid joint, tetanus, or cervical rheumatism. Dropping of head and neck might suggest paresis, mechanical injury to the levator muscles or cervical ligament, extreme debility, or prostration from a profoundly depressing fever or poisoning. Inability or indisposition to back, might indicate sprain or fracture of the back, ankylosis, laceration of the sub-lumbar muscles, paresis, cerebral or spinal inflammation, softening or other lesion, tetanus, laminitis, dislocated patella or certain other affections. Swaying or unsteadiness in walking or turning would similarly suggest sprain or fracture of the back, paresis or other nervous and locomotor injuries. The soliped with peritonitis arches the back and draws the hind feet forward under the belly, with impacted colon or obstruction to urination he will often stretch with fore limbs advanced and hind limbs retracted. The mode of decubitus may be significant. With peritonitis, enteritis, metritis or acute nephritis or hepatitis the soliped lies down slowly and with caution; with spasmodic colic he throws himself down as if reckless of possible injury. Lying well up on the costal cartilages and side of the breast bone suggests a slight affection of the air passages, lying on the side, disease of other parts. Rolling on the back may indicate simple intestinal spasm, but also blocking by intususception, impaction,

volvulus or otherwise. Sitting on the haunches may suggest a similar trouble or it may imply ruptured stomach or diaphragm. The dog may sit on his haunches in health, or with dyspnoea in acute affections of the respiratory organs, asthma and heart affections. Decubitus on the belly with hind legs extended backward, may imply paraplegia, or acute inflammation of the abdominal organs. Lying with the nose in the flank or turning the head toward the flank, though a normal position of rest, often indicates abdominal suffering. Turning of the head to one side may, however, suggest injury, spasm or rheumatism of the cervical muscles, or disease on the corresponding side of the brain. Animals, at liberty, lie more frequently on the side on which the heaviest internal organs are lodged, thus ruminants, pigs, and dogs rest on the right (the side of the liver), though in cattle with a heavily loaded rumen the condition may be reversed. Decubitus on the abdomen, with the limbs extended and abducted implies profound nervous disorder or shock.

Habitual decubitus often indicates severe suffering in legs or feet. Resting one limb more than another implies injury to that limb. Standing with the pastern of one limb more upright than the others has the same meaning. Extension of one fore foot in advance of its fellow with flexion of the pastern and fetlock denotes suffering in the posterior part of the foot or in the flexors. Flexion of carpus and fetlock without advance of the foot probably bespeaks injury to shoulder or elbow. Inability to bear weight on the fore limb, without knuckling at the knee, should call for examination of the olecranon and joints, especially the elbow. Inability to extend the carpus should lead to investigation of the flexor muscles and tendons, the joints and the heel. Movement of the hind limb without flexure of the tarsus would suggest injury to that joint, the stifle, or the flexor metatarsi tendon. Inability to extend stifle and hock should demand examination of the tendo-Achilles and os calcis, of the triceps extensor cruris and of its nerves.

Atrophy of a muscle or group would require scrutiny of its tendons and its nerve and blood supply.

More precise indications of injury of the locomotor system must be found under surgery.

After posture, the general or constitutional disorder may claim

attention. Is the illness acute or chronic? Is fever present? Has the animal had a rigor? Does the coat stare in patches (along the spine) or generally? Is there perspiration? Is there full, clear, somewhat congested eye (sthenic), or drooping lids over a dull brownish sclerotic (asthenic)? Are the lower parts of the limbs and other extremities cold, and the roots of the horns or ears hot? Is there significant heat and dryness of the muzzle (ox), nose (dog), snout (pig), palmar-pad (carnivora), hoof (solidungula, bisulcates), bill and digits (birds)? Has the mouth the hot burning feeling of fever? Finally is the temperature as indicated by the thermometer abnormally high? To estimate this with any degree of certainty one must be well acquainted with the normal temperature.

Normal temperature. As taken indoors under ordinary conditions, the normal temperature taken in the rectum may be: Fowl, 107° — 110° F.; swine, 103° — 106° F.; goat and sheep, 103° — 104° F.; ox, 100° — 102° F.; dog, 99° — 100° F.; horse, 99° — 99.6° F. Ranging in the fields, at work, or on forcing or stimulating feeding, it may be 1° higher than when at rest indoors. A whole herd may be raised 2° by a three miles drive in warm weather. In our summer heats a rise of 1° is common. In nervous animals any change in management may raise the temperature, for example, 1° to 2° after failure to water at the usual time, or from retaining the milk in the udder when the milker had been changed. Young animals are normally $.5^{\circ}$ to 1° warmer than old ones, though more sensitive to the action of cold. Half starved animals, when put on abundant and nutritious food may have a rise of 1° or more. Females in heat, in advanced pregnancy and at the time of parturition are usually 1° to 3° above the natural temperature. Among the agencies lowering temperature are: Cold (1° to 2°), sleep (1° to 2°), rest, starvation, alcoholic and other circulatory stimulants which fill the cutaneous capillaries and thereby cool the whole mass of blood; suppression of insensible perspiration (retention of waste matters) as by varnishing the skin which lowers the temperature to 25° ; purgatives and diuretics (1°); certain drugs like antipyrine, acetanilid, etc., which act on the heat producing centres and retard metabolic changes.

Temperature in disease. Comparative temperatures should

be taken at the same hours on successive days, bearing in mind that the morning temperature is usually slightly lower and the evening one slightly higher. Where possible both morning and evening temperature should be taken. With elevated temperature, repeat sooner to see that it is not transient. A transient rise of 1° to 2° is unimportant. A permanent rise of 2° or 3° indicates fever. A sudden additional rise of several degrees in the progress of fever is grave. A persistence of the high evening temperature to morning shows aggravation. A persistence of the low morning temperature to the evening bespeaks improvement. A sudden extreme fall to much below the normal (4° or 5°) indicates collapse. This is usually attended with other symptoms of extreme prostration and sinking. A sudden considerable fall to near the normal, without untoward attendant symptoms, may indicate a *crisis* and a more or less speedy improvement may be hoped for. This sudden fall often attends the period of eruption of certain exanthemata, as cowpox, horsepox, sheepox, aphthous epizootic, etc. A sudden extensive fall of temperature may result from some transient accidental cause, as a prolonged deep sleep, a hæmorrhage, the removal of constipation, or of enuresis. A sudden rise may supervene on such suppressed function or other cause of nervous irritation or on toxin poisoning, but it does not persist more than twelve or twenty-four hours after the cessation of the morbid cause.

A rise of 10° or 12° above the normal standard is usually promptly fatal.

A continued high temperature indicates persistent disease, and a considerable rise during defervescence implies a relapse and in the absence of any error in diet or nursing is grave.

Pulse. Before the introduction of the clinical thermometer, the indications furnished by the pulse were held to be of the highest value. Though largely superseded by the usually more reliable thermometer, yet they should not be discarded, but employed as symptoms corroborative of the thermometric indications. In many cases the pulse will furnish criteria, when in the absence of fever, the heat of the body will tell of nothing amiss. This is especially true of diseases of the heart, of the large blood vessels, and of the nervous system, and in cases of poisoning. For special indications furnished by the pulse, see diseases of the heart.

Respiration. The morbid activity or inactivity of the respiration, its modified rhythm, the pathological significance of the altered breathing sounds and of the superadded sounds, the indications furnished by percussion, palpation, mensuration, succussion, sneezing, snorting, yawning, cough, moan, grunt, stertor, discharge, etc., afford material of inestimable value to the diagnostician. See under diseases of the chest.

Skin Symptoms. The erection of the hair of carnivora in rage or fear implies a profound nervous disturbance, and a similar erection (staring coat) in the larger herbivora especially, implies a corresponding nervous disorder, due, however, to a different cause—fever. The pallor and coldness of (white) skin and extremities, the retrocession of blood toward the internal organs, the contraction of the involuntary muscles of the hair bulbs, the sense of cold, and the actual shivering all come from the fundamental nervous disorder. The loss of lustre and gloss in the hair and the dryness, rigidity and lack of mobility (mellowness) of the skin imply lack of nutrition. The mellow feeling of the skin under the pressure of the finger, soft and yielding by reason of the lax connective tissue and fatty layer in the thrifty animal, is in marked contrast with the dry, hard, tough, unyielding hide firmly adherent to the parts beneath (hidebound), which denotes the unhealthy or unthrifty animal, or from the thin, attenuated, mobile, bloodless skin of the debilitated subject, the victim of lung, liver, or intestinal worms. In sheep in parallel conditions there is a lack of *yolk* in the wool, which is dry, lusterless and brittle and often flattened (clapped) on the skin. In fowls ruffling of the plumage indicates the nervous disorder and chill. The skin may be scurfy in conditions of low health or in connection with the presence of vegetable or animal parasites. Ringworm has excessive scurf, and tends usually to a circular form, and to complete shedding of the hair from the affected spots. The hairs split up before dropping. In acariasis there may be scurf, scab, abrasion and sores of many kinds, but the outline is not necessarily circular, nor strictly limited, isolated hairs remain even on the bare patches, and itching is extreme as shown by the movement of the body and especially of the lips or foot when the part is scratched.

The hair may be freely shed during convalescence from debili-

tating diseases, a condition that must not be confounded with the yearly shedding of the winter coat and the moulting of birds, which is a perfectly normal process. Yet even the spring shedding and the growth of a new coat makes a great drain on the system, and must always be taken into account as a probable cause of derangement of health.

The lesions of the skin in the different cutaneous affections must be remanded to the special chapter on skin diseases. The following however may be named as having a general bearing.

Emphysema may be due to a local wound, (elbow, trachea, rib); it may indicate black quarter, or it may occur sub-cutaneously in cattle without marked impairment of health.

Anasarca, from diseased blood, heart, liver or kidneys, is denoted by swellings, often painless, or a general infiltration which pits on pressure. It often shows primarily in the lower parts of the hind limbs. Warty looking elevations must be carefully discriminated, having in mind primarily papilloma, tubercle (grapes), actinomycosis, condyloma (in dogs), cancer, melanosis. The secretions of the skin (sweat, sebum) may be suppressed, or in excess, producing at times a special odor, as in thrush and canker of the horse, cowpox and sheep-pox, and rheumatism. Before death the cadaveric odor may be marked, and attracts crowds of flies to the victim.

Facies. The countenance may be expressive. Between the bright, full, clear, prominent eye of health, and the dull, sunken, lifeless semiclosed eye of serious disease the contrast is extreme. The drooping lids (ptosis) may be paralytic and even unilateral, in which case drooping ear, and flaccid lips and alæ nasi complete the picture. With paralyzed lips there is usually drivelling of saliva, and dropping of half chewed morsels in the manger and stall. The eye may show dropsical lids in kidney or liver disease and in anæmic conditions like distomatosis in sheep. It may show the upper lid bent at an angle in recurrent ophthalmia of solipeds. The mucosa may be red in ophthalmia, yellow in jaundice, dusky brown in Southern cattle fever, anthrax, cerebral meningitis, and other fevers attended with destruction of red globules and liberation of their hæmatin. The pupils may be all but closed in internal ophthalmias, or widely dilated and irresponsible to light in amaurosis. The iris may lack its normal

lustre or may be distorted or torn in various ways from adhesions. Opacities of the cornea, lens, or vitreous may be recognized.

The facial muscles may be flaccid and devoid of expression in palsy and prostrating diseases; they may be firm, giving the bright, intelligent look of health; or they may be painfully drawn in the agonized expression of spasmodic colic or enteritis.

Nasal Mucosa. The pituita is bright red in sthenic fevers, simple acute coryza, strangles, laryngitis, and inflammation of the larger bronchia. It assumes a violet hue in capillary bronchitis, pulmonary congestion, glanders and petechial fever. Petechiæ appear in the lastnamed affection, and in a number of bacteridian diseases, such as anthrax, swine plague, hog cholera, the red fever of swine, etc.; a yellow tinge is shown in jaundice. Millet-like or pealike nodules, or elevated patches, and ulcers show in glanders and may be felt by the fingers. In cattle, hard, millet-like nodules appear in a chronic coryza with hypertrophy of the mucosa. The orifice of the lachrymo-nasal duct, seen in the horse on the floor of the chamber at the junction of the mucosa with the skin of the false nostril and in ass and mule on the outer ala near the upper commissure, is sometimes plugged with inspissated mucus. Among other lesions of the nasal chamber may be named polypi—soft and calcareous—thickening and obstruction in purpura hæmorrhagica, osteoporosis and hypertrophy of bone, and parasites—pentastoma denticulata (in the horse and dog), and the larva of the oestrus (in sheep and buffalo). Disease of the upper molars and abscess of the fronto-maxillary sinus may be manifested by swelling beneath and on the inner side of the eye, foetid discharge from the nose, and obstruction of the air current. Dullness on percussion will show the filling of the sinuses. These conditions must be carefully differentiated from actinomycosis, sarcoma and other morbid growths in the same situation.

Costiveness with fœtor and lack of the normal color in the stools may suggest *liver torpor* or inflammation, while fatty stools may suggest pancreatic disease. The uneasy movements of colic, should lead to a careful investigation of the chylo-poietic organs (see digestive organs). Weakness of the hind parts, tenderness of the loins, and altered condition of the urinary discharge should demand a close enquiry into the state of the *kidneys* and *bladder*.

Satyriasis or nymphomania would suggest disease of the *generative organs* or the nerve centers that preside over them. The same is true of impotency, sterility and abortion.

In eruptions on the skin (erythema, eczema, pustule, squama) a cause may be found in the local action of heat, friction, or other direct irritant, but in the absence of any such manifest cause, an enquiry should be made into the functions of sanguification, digestion, urination and the action of the liver. It may further suggest parasitism (ringworm, phthiriasis, fleas, acariasis, verminous disease, etc.)

Symptoms of *nervous disorder* are too numerous to be here traced to local lesions. Motor paralysis of one limb may, however, suggest injury to its motor nerves, to the same side of the spinal cord, or the opposite half of the cerebrum. Paraplegia almost always indicates injury to the cord. Sensory paralysis of one side may depend on disease of the opposite corpus striatum. The animal moves in a circle when a tumor (coenurus in sheep) exists in the roof of the lateral ventricle presumably pressing on the ganglia on its floor. An animal rolls on its axis when there is a lesion of the median cerebral peduncles, of the supero-external portion of these peduncles, of the posterior part of the encephalon, or of different parts of the hemisphere. Amaurosis suggests disease of the corpora quadrigemina. Loss of coördination of muscular movement usually implies some lesion of the cerebellum. Vertigo may imply disease of the encephalon (congestion, anæmia, inflammation, dropsy, hæmorrhage, tumor, abscess); it may be disease of the internal ear; it may be digestive disorder connected often with cryptogamic poisoning; it may be heart disease with obstruction of the jugular veins; it may be parasites in the nasal sinuses; or it may be disease of the eye. Coma occurs in most congestions and pressures on the encephalon, and like vertigo, in poisoning by alcohol, solanine, monoxide of carbon, etc. In acting on any ganglionic center the agent may, according to its degree, operate positively or negatively, producing spasms, or paralysis as the case may be. As in the case of other visceral affections the specific disease must be referred to for particular symptoms.

For the more precise points in diagnosis, including chemical, physical, electrical and instrumental methods, etc.; the reader is referred to the special diseases.

PROGNOSIS.

Definition. Demands on the veterinarian, the question of economy. **Basis of Prognosis.** Cause of the illness, internal or external, vital or non-vital organ, enzootic, fatigue, infection, in one or two symmetrical vital organs, regular or irregular in its course, persistence, relapse, complications, effect of treatment, appetite, temperature, pulse, breathing, youth, age, debility, previous disease, breeding, climate, season.

Prognosis is a more complicated question for the veterinarian than for the physician. The latter must pronounce on the malady, whether it is likely to follow a regular or irregular course, whether it will last short or long, whether it will be curable or incurable, and if curable whether recovery would be complete or partial. For the veterinarian there is in addition the question of economy. The veterinary patient is, as a rule, of value, only if he can be rendered sound, and a partial recovery may be even worse than a fatal result, since the subject remains as a ruinous charge on his owner. The veterinarian must pronounce on the prompt and perfect curability of the case, on the outlay that will be requisite for treatment, on the depreciation which will be entailed on the patient, and whether, in certain lesions that do not harm the carcase, it would not be more judicious to butcher the subject. The physician is expected to do the best he can for life and health, and even a very imperfect recovery brings him a mead of gratitude. The veterinarian on the other hand must be an expert not only on disease, but on animal values, and if his treatment, however skillful it may be, results only in the prolonging of the life of a useless animal, the owner may charge him with imposing upon him an unnecessary outlay. The soundest judgment and highest skill are often necessary to secure the interests of an employer in such circumstances. In certain cases the recommendation to destroy is of much more value to the employer than the most skillful, and partially effective curative treatment. On this basis, the reputation of a skillful man may be securely built. He can deceive no one if his prediction of recovery is not justified, while if he advises destruction and the patient recovers, he is at once discredited.

To give a sound prognosis the practitioner must have a thorough knowledge of pathology, he must have acute powers of

observation, and he must be quick to appreciate every point that makes for or against the patient in the particular case.

The *causes* of the trouble must be carefully considered. Are they transient or permanent? Are they removable or irremovable? Are they external or internal? As a rule an internal cause is the more redoubtable. Some lesions are necessarily fatal, as a needle penetrating the heart or an attack of rabies or milk sickness. Is the cause an enzootic one? If so can the patient be removed from the locality? Is it a fatigue fever or an infectious one? Is it a simple inflammation or an infecting one? The latter are usually much more grave. In case of contagious disease, can its propagation be prevented? Is it of a fatal or non-fatal type? Is it situated in a tissue favorable to a fatal extension (anthrax in lung), or in one unfavorable (anthrax in the tip of the tail)? Disease in a single vital organ like the heart is necessarily much more grave than is one of a symmetrical pair (kidneys, lungs), one of which can carry on the functions. The regular progress of the disease and especially an uninterrupted improvement, following on a critical perspiration or urination, is a good prognostic sign, whereas unevenness of temperature, pulse and respiration, with temporary aggravations of the general symptoms, should demand a less hopeful prognosis. The persistence of the malady is also an unfavorable indication. A relapse after partial recovery is a serious indication unless due to some obvious and easily removable cause, and unless the former convalescent condition is speedily restored on its removal. A complication is a serious indication whether it consists in an embolism or new centre of the same disease, or the supervention of a second disease upon the first. The system has just so much more to contend with and the very supervention of the second focus or malady argues a special susceptibility, debility, or lessened power of resistance.

The prompt success or entire insuccess of treatment proves valuable as a guide.

The preservation of appetite, the slow, uniform descent of the temperature, and the improvement of pulse and breathing are among the most valuable indications.

Something may be deduced from the condition of the patient. If very young or old, debilitated by overwork, bad or insufficient

food, previous disease, or any other cause, the prognosis is less hopeful, as it is also as a rule, during gestation, in the parturient state, or if abortion ensues. A hereditary predisposition to the malady in question is equally unfortunate.

Climate may be an important factor. Thus liver diseases are far more to be dreaded in a damp tropical or semi-tropical region, and rheumatism and catarrhal affections in winter and in cold northern localities. Acclimatization should also be considered. The bovine animal, raised on the Gulf Coast is likely to make a good recovery from Southern Cattle Fever, (pyroplasmosis) while the northern beast would almost certainly die.

All in all the question of prognosis cannot always be judiciously decided at a first visit, and for the sake of his own reputation, it is well that the practitioner should give only a qualified opinion at first until he can certify himself as to the probable outcome of the disease.

PROPHYLAXIS. PROPHYLACTICS. PREVENTION.

A test of public sentiment. Soil. Water. Exposure. Buildings. Local hygiene. Breeding. Diet. Work. Harness. Ventilation.

With advancing knowledge of veterinary medicine the subject of prophylaxis is steadily assuming a more important place, and especially in the classes of enzootic and epizootic diseases. Indeed for the fatal infectious diseases of animals one can fairly estimate the medical intelligence of the people by the extent to which therapeutic treatment is still allowed. With economy as the great central object of veterinary medicine, the problematic recovery of the few can never balance the assured preservation of the many. But this subject belongs to contagious diseases to which the reader is referred.

In enzootic affections, improvements in soil, water, exposure, buildings, and other local unhygienic conditions, are the final ends to be sought, according to the particular nature of the prevailing disease.

So in sporadic diseases the correction of faults in breeding, hygiene, diet, water, work, harness, exposure, buildings, ventilation, etc., are called for in different cases as will be noted under the individual diseases.

THERAPEUTICS. TREATMENT.

Definition. Mechanical and Medicinal Therapeutics. Adaptation to each case of disease.

The ultimate object of all medicine is to prevent disease, or when it cannot be prevented, to cure. The term therapeutics covers all measures applied with curative object. Therapeutics are naturally divided into **Mechanical and Medicinal**. To mechanical therapeutics pertains the whole domain of surgery. Medicinal therapeutics has to do especially with internal medicine. Each of them, however, encroaches more or less on the other. Modern surgery is essentially aseptic, or antiseptic, and antisepsis is secured by medicinal agents. In medicine when cups are applied we adopt an essentially mechanical treatment. Both methods then must remain open to physician and surgeon. Another and no less important branch of treatment which is open to physician and surgeon alike is diet and general hygiene. The same care must be given to the use of these in the treatment of disease as in its prevention, and in many cases a judicious use of these may almost entirely obviate the necessity for medicine.

It would be useless to enter here into the subject of therapeutics. Suffice it to say that the choice of a system and of individual agents must be determined by the particular conditions of the case, its cause, and nature, the strength, vigor, and genus of the patient, the organ involved, the extent and stage of the disease, the existence of a relapse, or complication, and all other circumstances that would affect the action of the remedy. Specific statements must be made with the several diseases.

HYPERÆMIA. CONGESTION.

Definition. Forms, active—arterial, passive—mechanical—venous. Determination of blood. Causes of active congestion. Vaso-motor nerves. Lesion of spinal cord; or of sympathetic nerve. Reflex irritation. Central cause. Physiological hyperæmia. Medicinal hyperæmia. Microbian (toxic) hyperæmia. Arterial obstruction. Thrombus, tumor. Cold, chill. Removal of pressure. Cardiac hypertrophy. Symptoms, bright red color, swelling, dropsy, migration of cells. Rise of local temperature. Tenderness. Altered function. Causes of passive congestion. Obstructions in the lungs, heart, veins. Diminished force of circulation from age, debility, arterial disease, distance from the heart, decubitus, vaso-motor disorder. Gravitation—hypostatic congestion. Tumors. Paresis. Symptoms. Cyanosis. Distended veins. Coldness. Transudation—watery. Hæmorrhage. Thrombus. Hyperplasia. Atrophy. Postmortem lesions. Treatment. Remove cause. Correct injurious gravitation. Correct any fault in blood pressure. Derivation. Constrict or support part. Massage. Electricity. Improve general health.

Definition. An excess of blood in a part. It is distinguished from inflammation by the absence of that tissue reaction, which leads to or constitutes the special phenomenon of that morbid process.

Hyperæmia is divided into **active or arterial** and **passive, mechanical or venous**. A capillary form has also been described but usually capillary congestion is seen in both the arterial and venous types.

I. Active or Arterial Determination of blood. In this form the arteries are dilated under a direct nervous influence. *Causes.* In all the regular functions of the body, the flow of blood is under the direct control of the vaso-motor nerves which proceed from the spinal cord, through the branches of the sympathetic to be distributed with blood vessels. The hard pulse of pleurisy is due to rigid contraction of the constrictor muscles under the action of the vaso-motor nerves, and the blush of shame is due to their relaxation. Some claim an active dilatation of the arterial muscular coats, others look more simply upon the dilatation as a mere yielding of the coats under the blood pressure, when the constrictor muscles are relaxed. This vaso-

motor paresis may be induced : 1st, by any lesion of the spinal cord. 2d, by the cutting of a sympathetic trunk, that of the abdomen, for example, which leads to active congestion of the abdominal viscera, or the section of the cervical sympathetic which leads to watering of the eye, sweating, congestion, and scabbing on the corresponding half of the face. 3d, by reflex irritation through the sensory nerves, as in congestion through friction, heat or cold to the skin, or that resulting from excessive use of an organ such as the mammary gland. 4th, by causes acting directly through the mentality as in emotional blushing or the facial congestion of violent rage.

Physiologically we see the operation of this nervous control in the congestion of the gums during dentition, of the salivary glands during mastication, of the stomach and bowels during digestion, of the womb during gestation, of the mammæ at parturition, and of erectile organs in copulation.

Medicinal agents act in the same way, opium or alcohol producing active dilatation, and belladonna and ergot causing active contraction of the arterial walls.

Microbian poisons may act in the same way, tuberculin and a number of others causing active dilatation.

The obstruction of one artery by thrombus, tumor, or ligature, causes increased tension in the collateral branches coming off just above and an active congestion in the parts to which these are distributed. While this is directly due to increased local pressure, it is also an instance of the lack of balance between the blood pressure and the resistance in the vascular walls. In this case there is increase of pressure, in the other a diminished resistance.

If there is a superficial anæmia, as from cold or chill, there is of necessity an internal hyperæmia. This contributes to the production of internal congestions and inflammations, though the seat of election of such inflammation is usually determined by the nervous sympathy between the part chilled and the deeper organ affected.

Another cause of congestion is the lessening of pressure by the parts surrounding the vessel. Thus in cupping, there is prompt cutaneous congestion, and a similar result occurs in pericardium, pleura, or peritoneum on the withdrawal of the liquid of hydro-pericardium, hydro-thorax or ascites.

Another cause of congestion is found in hypertrophy of the heart and increased force of the blood flow (blood tension). In such cases those organs become congested in which there is some previous debility or disease of the blood vessels.

Symptoms and results. The symptoms are a bright vermillion redness, tension or swelling, heat and tenderness. Pulsation is stronger in the vessels leading into the part, secretions tend to increase but may give place to a serous effusion or hæmorrhage. The bright redness is attributed to the rapid circulation of the red globules which have not time to give up their oxygen to the tissues. It is sharply circumscribed where the affected arterioles have no free anastomosis with those of neighboring parts, diffuse where anastomosis is abundant, and when on the skin it is liable to rise in knots or buttons as in urticaria. When pressed the redness entirely disappears unlike the redness of inflammation.

The swelling may be due to the simple turgescence of the blood-vessels, but also often to transudation of serum as in and around the cow's udder at parturition. The occasional migration of globules, and their escape through minute lacerations in the vascular walls add alike to color and turgescence.

The elevated temperature (rising sometimes 3° C.) in the congested area, is attributed to the more active circulation, and Schiff prevented its appearance after section of the cervical sympathetic, by tying the carotid and vertebral arteries on the same side.

The tenderness of the congested parts varies inversely as the looseness of texture and the facility for swelling. It may be scarcely perceptible in the mammary region, and intense under the horns or hoof.

The functions in the congested organ are often seriously interfered with, secretions appearing in excess or entirely altered. When the congestion lasts it may cause hypertrophy, induration or hyperplasia, these are however rather sequels than lesions of the condition. Simple congestion is usually quite transient, and if prolonged, often merges into inflammation.

II. Passive or Venous Congestion. In this there is no excess of blood entering the part, but the regular supply is delayed in the veins by some obstruction, and these vessels and, later, the capillaries are gorged with black blood.

Causes. 1st, Mechanical obstruction to the outward flow of blood as in the case of disease of the lungs hindering the flow of blood from the right heart ; disease of the right heart allowing a reflux of blood into the veins ; or pressure by tumors or otherwise on the great or small venous trunks. If in the heart or lungs the whole systemic venous system becomes the seat of passive congestion ; if in a single venous trunk then only the parts the venous radicles of which are tributary to this. We find examples of this in phlebitis, in compression by the swellings of strangles, in the result of a bandage or ligature tied round a limb at some distance from its extremity, and in the compression of the iliac veins by a gravid womb.

2d. Diminished force of the blood current in the veins, as from old age or great debility and especially from weakness of the heart's action. Also from disease of the arterial coats which impairs their tenacity. The force being too weak to propel the blood actively through the capillaries and veins, it becomes unduly charged with carbon dioxide and other products of tissue waste, so that nutrition suffers and the walls of the capillaries lose their vital force. This condition is aggravated in the hind limbs by the distance from the heart, and the dependent position, and in decubitus by the compression of the vessels of the limbs. Also by injuries to the vaso-motor nerve supply as œdema appeared in the hind limb after tying of the femoral vein in animals the abdominal sympathetic of which had been cut, but not in animals in which this nerve was left in its normal condition (Ranvier).

3d. Gravitation in weak states of the circulation must be looked upon as a cause of venous congestion. This is seen in the examples of hypostatic congestion and œdema seen in the lungs and other internal organs in low conditions and in advanced stages of debilitating diseases, and in certain cases of stocking of the limbs in horses.

4th. Valvular insufficiency of the left heart and tumors or aneurisms interfering with circulation through the aorta, cause passive congestion of the pulmonary veins and œdema of the lung,

5th. Tumors and diseases of the liver determine passive congestion of the portal system and ascites.

6th. Passive congestion is very liable to take place in an organ

the functions of which are impaired as in a paralyzed part. In this the hyperæmia may start in the capillaries and extend to the veins or even to the arteries.

Symptoms and results. If on a mucous membrane or white skin the color becomes dark red, or violet (cyanotic) with evident distension of the capillaries and veins, the latter of which may stand out as knots or cords, there is an appearance of swelling or enlargement and sometimes coldness of the part. Soon the watery part of the blood transudes in excess, constituting dropsy, with increased swelling and pitting on pressure. On the mucous surfaces it determines an abundant serous secretion. The color is deepened by the escape from the vessels of red globules as well as white. The transudation contains little albumen and only exceptionally fibrine. In connection with the marked deoxidation and high carbonisation of the blood, the nutrition of the part is largely arrested together with the functions, secretory, motor or otherwise. The imperfectly nourished vessels may give way, leading to hæmorrhage, or nutrition may be definitely arrested producing moist gangrene or ulceration. Sometimes a thrombus is formed in a congested vein. The changes in the affected organs depend much on the degree and duration of the hyperæmia. If slight and lasting it causes permanent induration and thickening, from connective tissue hyperplasia, as frequently seen in the hind limbs of the horse. In case of blood transudations the altered coloring matter gives the various shades of gray, brown or black. If long continued the organ may shrink and atrophy occur from defective nutrition and contraction of the fibrous hyperplasia.

In making post mortem examinations mistakes may be made through the occurrence of changes after death. Thus a hyperæmia which was quite considerable during life may virtually disappear through the contraction of the arterial and capillary coats forcing the blood on into the veins. A minute point of extravasation here and there may be the only macroscopic lesion left. Again a marked venous and capillary hyperæmia in a dependent part of the body or of an organ may be entirely due to hypostatic conditions, the blood having settled into the lowest part of the vessels since the death of the animal. To avoid this source of error one must always carefully note the position of the carcass

after death. Under other circumstances the superficial veins and capillaries may fill up with blood through the occurrence of decomposition and the evolution of gases in the internal cavities, which empty the splanchnic and parietal vessels by compression.

Treatment. The general principles of treatment may be stated thus: 1st. Remove the cause of the hyperæmia if possible, especially any mechanical cause; 2d. Secure the influence of gravitation in favor of the return of blood to the heart; though not so available in animals as in man, it is of great value in congestion of the head, ears, tail, and to a less extent of other parts; 3d. Correct any fault of blood-pressure, excess or deficiency, which may act so as to cause active or passive hyperæmia; 4th. Establish derivation by cupping, leeches, fomentations, pediluvia, sinapisms, etc.; 5th. Apply cold, astringents, bandages, to empty the hyperæmic vessels, or kneading, rubbing or electricity, to hasten the flow of blood; 6th. Improve the quality of the blood and general health, in plethora by low diet, purgatives and diuretics, in anæmic or debilitated conditions by iron, bitters, nourishing food, fresh air, sunshine and exercise.

It is especially important to check passive congestion in febrile diseases, and mechanical congestion at an early stage of its progress (Roberts).

INFLAMMATION. PHLOGOSIS. PHLEGMASIA.

Definitions. Relations to active hyperæmia. Redness. Heat. Pain. Swelling. Forms : in vascular tissues : in non-vascular. Changes in tissue elements. Death of cells. Cloudy-swelling. Granular degeneration. Cell proliferation. Karyokinesis. Embryonic cells. Amœboid functions. Migration of leucocytes. Red cells escaping. Changes in innervation. Vaso-motor disorders. Fever. Changes in circulation. Contraction of capillaries, dilatation, rapid flow, tardy flow, stasis, oscillations, thrombus, collecting of white globules in periphery of current, migration of leucocytes, blood plates, and red globules, massing of red globules, exudation, softening of the capillary walls, nutrient artery more rigid and transmits more blood, heart contracts more forcibly, increase of fibrine, increase of waste products. Buffy coat, physiological causes. Haemolysis. Microbes. Pto-
maines. Toxins. Chemiotaxis. Phagocytosis. Polynuclear and mononuclear leucocytes. Exudates, unlike dropsies. Mucous exudate. Serous exudate. Fibrinous exudate. Blood exudations. Croupous exudation. Chyliform exudate. Results and products. Resolution. Deletescence. Metastasis. New formations. Suppuration. Pus microbes. Pus. Healing by 1st intention. Healing by 2nd intention, granulation. Granule corpuscles. Interstitial neoplasia. Degenerations in lymph. Fatty degeneration, melanotic. Softening. Ulceration. Gangrene.

Inflammation has been variously defined as "perverted nutrition," as a "protective reaction of the organism against irritant agents" and in other terms that express at once too much and too little, without actually defining the morbid process. Older definitions dealt with the manifest disorders of circulation, of innervation or of tissue change too often exalting the importance of one set of changes at the expense of another and thus giving in the main a one sided view of the morbid process.

Some modern bacteriologists are inclined to refuse the title to any morbid process that is not caused by the presence of microbes or their toxic products. To them the changes occurring in an aseptic wound or in a simple fracture in process of healing are purely reparatory and partake no more of the nature of inflammation than do the developmental changes in the growing embryo. While to a large extent true, this exclusive view implies exceptions, since if the chemical poisons derived from the bacteria can develop inflammation, the same must be admitted as possible for chemical irritants drawn from other sources,

As a matter of fact inflammation, occurring as it does in very different tissues, vascular and nonvascular, fibrous, cellular, parenchymatous, etc., and in connection with a great variety of irritants, must be held to include a large group of morbid processes, bearing to each other a strong family relationship and resemblance, and yet differing in many important details. Each irritant (heat, cold, electricity, chemical irritant, incised, punctured, lacerated or contused wound, rupture, fracture, foreign body, parasite, microbe, toxin, etc.,) has its own special character and mode of irritation; each tissue has its own special method of succumbing or reacting and its own amount of blood supply; and each system and organ has its own native or acquired power of resistance and reaction.

Inflammation agrees with active hyperæmia in the tendency to dilatation of the vessels and an increased flow of blood to the part or if the irritated part is nonvascular like the cornea or articular cartilage, then to the parts adjacent. It differs, however, in the more active cell proliferation, and in the nature of the liquid transudation which is richer in albumen, fibrine, cells and phosphates. Abstractly the inflamed part retains very active vital processes, trophic and exudative, but these, are largely changed from the normal and are, it is claimed, perverted, yet they preside over the processes of cell growth and decay, the removal of injured or useless tissue, and later, over the building up of new material, and repair of loss. Active hyperæmia on the other hand is mainly a circulatory disorder, and when it advances so as to determine changes in the cells and tissues it is held to have merged into inflammation.

The term inflammation (from *inflammo*, I set on fire), is suggestive of the local heat of the inflamed part, just as fever (*febris*) indicates an elevation of the temperature of the body at large. Celsius enumerated the features of *robor*, *calor*, *dolor* and *tumor* (redness, heat, pain and swelling) which have come down to our own time as at least suggestive of inflammation. But any diagnosis, based on these alone, would be today woefully inadequate. Redness occurs in the transient blush, heat in the febrile state, though no inflammation can be recognized, pain is present in neuralgic and other nervous affections, and swelling in dropsy and tumor. On the other hand redness is entirely absent, for a time,

after the onset of inflammation in nonvascular tissues (cornea, articular cartilage), the heat of the inflamed part may be actually lowered when there is much exudation around the capillary vessels and lessened flow of blood, pain may be absent in some circumscribed inflammations of the lungs, and swelling is not at first visible in the inflamed cornea or compact bony tissue. These phenomena which are so common in inflammation and, in general so characteristic of it, cannot therefore be accepted as infallible evidence of its existence, nor can their absence be held as absolutely implying its nonexistence.

Forms of Inflammation. This morbid process might be divided almost indefinitely according to the organ invaded, the cause, and type, yet it will be more convenient to deal with it generically and notice **inflammation in nonvascular and vascular tissues** respectively, and the different types of **granular degeneration, exudative inflammation and croupous inflammation**. It will be requisite further to notice an **acute** and a **chronic** type.

By dealing first with the changes in the anatomical elements of the tissues and in the innervation, we shall virtually cover the phenomena observed in nonvascular tissues, and later the changes in connection with the circulatory system will give the additional characteristics of inflammation in vascular tissues.

CHANGES IN THE TISSUE ELEMENTS.

Death of cells and tissue. By the application of an irritant (acid, heat, etc.,) a certain thickness of tissue with its enclosed cells is killed, and a thin layer of necrosis is usually produced. This does not constitute inflammation, but it acts as a foreign body, often septic, in producing inflammation in the parts adjacent.

Cloudy Swelling, Granular Degeneration. This may occur in the inflamed area surrounding the necrosed tissue in the seat of a burn or other injury, it is exceedingly common in the cells of inflamed parenchymatous tissue (liver, kidney); in the muscle of the heart, in the gastro-intestinal mucosa, in febrile affections and in poisoning with arsenic, phosphorus, or mineral acids. The gross appearance of the tissue is that of swelling, with a dull

grayish color and a loss of its normal translucency. The cells of the affected organs are seen under the microscope to be filled with small albuminous granules which may be so abundant as to completely conceal the cell structure. The granules are insoluble in ether, but disappear under acetic acid. This condition of the cells is often associated with the exudative forms of inflammation.

Cell Proliferation and Change. In the nonvascular organ attacked by inflammation the multiplication of tissue cells and their resumption of amœboid movements is a constant phenomenon. Virchow insisted on the fundamental relation of the cell to the morbid process, and Goodsir and Redfern showed the rapid increase of the cells of articular cartilage in attacks of arthritis. There is first a sensible increase of the nucleus of the cartilage cell which shows a more extended and deeper staining in carmine or aniline; then by a special method of division (*karyokinesis*) the cell and nucleus divide in two; by a similar process these divide in four and so on in regular order. Meanwhile the cartilaginous substance becomes softened and finally dissolves and disappears, leaving in the place a mass of closely aggregated cells.

In the nonvascular transparent cornea, the membrane of Des-cement, the epithelium of serous membranes and in the epidermis a similar cell multiplication occurs, also in the lateral cartilages of the horse's foot.

To follow the indirect cell-division by *karyokinesis*, we must note the cell as a semi-solid mass, formed of protoplasm and nucleus, each having as its framework a network of exceedingly finer inter-crossing filaments, much finer in the nucleus than in the cell protoplasm. The nuclear filaments stain with hæmatoxy-lon and safranin, and are called *chromatin* threads. The intervening non-staining material is *achromatine*. The nucleus has a membranous envelope in two layers, of which the inner only stains. When about to divide two poles are formed in the cell protoplasm opposite to each other and near the nucleus, the filaments concentrating to the poles. The chromatin threads in the nucleus thicken, become convoluted, split and multiply, and draw into their substance the chromatin layer of the envelope. Next the chromatin threads form long loops directed toward an achromatine center or pole like a star, and this is followed by the progressive division of the star-shaped mass into two equal parts.

Finally they separate, together with the cell protoplasm, forming two daughter cells.

This cell proliferation under the action of an irritant is common also to the vegetable kingdom in which galls and tumors are formed in this way. It is a remarkable feature of these multiplying cells that they not only lose their power of developing the tissues in which they formerly lay, and have all their vital powers devoted to proliferation, but they acquire the amœboid power of their ancestors, the embryonic cells, which they further resemble in size. Indeed these cells are freely spoken of as embryonal cells, and the tissue formed by their massing together as embryonal tissue, and there is a widespread impression that they revert entirely to the form and characters of the embryonic cell. In some respects, however, they are unlike. The modified tissue-cell of inflammation presents a nucleus of horseshoe outline, or after division of the nuclei they together retain this semi-circular outline; it has the power of actively digesting the adjacent tissues as the embryonic cells do not, and again it does not possess the power of differentiation into widely different tissues as does the early embryonic cell. It may be called a reversion, in the direction of the embryonic cell, however, since it reacquires a number of its functions.

Migration of white blood cells. This is another, and in vascular tissues the main source of the great cell accumulation in the inflamed tissue. This process was observed by Waller in 1846, but was given its true importance through the later observations of Cohnheim. The migration takes place through the walls of the capillaries and veins only, and the migrating cells are largely of the poly-nuclear variety of leucocytes. These remaining adherent to the inner wall of the blood vessel may be seen to have a small portion of their substance projected through the wall and appearing as a small buttonlike projection on the outer side. This gradually increases, while the remaining portion of the cell on the inner side of the wall correspondingly decreases until the whole cell is lodged in the tissue outside the vascular wall. The time occupied in passing through is very varied. It may be wholly accomplished in half a minute, and again hours may be required for the complete passage of a single leucocyte. The explanation of this migration has been sought

in the supposed existence of stigmata (openings) in the vascular walls (Arnold), in the effect of the blood pressure within the inflamed vessels, in softening of the vascular walls and, in the contractility of the leucocyte which is strongly attracted by the presence of certain bacteria and other irritants (chemiotaxis). The migrated leucocyte assumes in the tissues the same habit as the altered tissue nucleus. It multiplies rapidly, assists in the solution and removal of the inflamed tissue, contests the ground with infective microbes (phagocytosis), and subserves the purpose of assisting in building up new tissue, or of degenerations.

Red Cells. The red blood globules follow the active current in the centre of the blood vessel, yet a few of these also become adherent to the softened walls and pass through them (diapedesis). When stasis of blood takes place in the vessels, they become packed more closely with red globules which then pass outward into the tissues in much larger numbers.

Changes in innervation. As shown under hyperæmia the vaso-motor system of nerves exert a potent influence on the circulation and is largely instrumental in bringing about circulatory disorders. The increase in the number and force of the contractions of the heart, and the rigid contraction of the walls of the arteries proceeding to an inflamed part, are distinctly the result of a reflex nervous action. The implication of the second eye when one has been violently inflamed from a mechanical injury is another example of this kind. The loss of power of the vaso-motor nerves is, however, even more characteristic. Experimentally the cutting of the cervical sympathetic or crushing of the superior cervical ganglion causes congestion and finally inflammation of the structures on that side of the head; the crushing of the semi-lunar ganglion similarly affects the abdominal viscera; and the cutting of the pelvic plexus, the structures of the hind leg. The contraction and dilation of the inflamed capillaries is largely a nervous phenomenon. A certain number of irritants, like warm water, mustard, or ammonia cause contraction, followed by dilation of the capillaries, while others like dilute mineral acids, alkalies, chloroform, or sodium chloride and sugar in concentrated solution produce dilatation at once. Some poisons act variously on different parts, eucalyptol causing dilation of the arteries and contraction of the veins, while cor-

rosive sublimate causes contraction of the arteries and dilatation of the veins.

So with certain microbial toxins. Introduced into the general circulation they produce active congestion or inflammation in the seat of colonization of the microbe from which they were derived, as witnessed in the use of tuberculin or mallein. Finally the chill and febrile reaction which attends on extensive inflammation is essentially a nervous phenomenon in its inception and progress.

Changes in the circulation. The usual changes in the bloodvessels of the inflamed part may be thus succinctly stated :

1. Contraction of the capillary vessels of the affected part and hastening of the current of blood through them.
2. The succeeding dilatation of the capillaries and the slowing of the blood stream, which still flows uniformly throughout the diseased tissue.
3. The flow of blood becomes irregular, at points tardy, and at others oscillating or even recoiling between the pulse beats when it has been forced into a vessel already blocked by coagulum.
4. In the still pervious vessels the red blood globules occupy the centre of the vessel where the current is rapid, while the white globules roll slowly along the inner surface of the walls, where the current is slow, and become adherent to the walls and stationary, while the general current rolls on. This is a direct abstraction of the white globules from the circulating blood and greatly favors the coagulation of the blood in the capillaries. The blood plates equally collect in the periphery of the vessel and escape.
5. The adherent white globules migrate in large numbers through the capillary and venous walls into the tissues. The red globules migrate to a less extent at first.
6. Small coagula form in the affected capillaries, forming minute red points which cannot be pressed out by the finger.
7. The red globules in the area of stagnation back of these capillary emboli adhere to each other by their flat surfaces and form rolls which pack into the vessel and are enveloped in a fibrinous clot.
8. The liquid part of the blood rapidly exudes into the tissues leaving the red globules relatively much more abundant in the liquid which remains inside the vessel.
9. The walls of the capillaries become softened and allow a readier transudation of liquor sanguinis, and escape of the globules through the walls of the vessels.
10. The arteries leading

to the inflamed part have their muscular coats more rigid and unyielding and transmit much more blood than the corresponding artery leading to the healthy part. 11. The heart is equally roused to more rapid and often more forcible contractions, which modify the pulse both in number and rhythm. 12. The circulating blood is found to have received a great increase in the fibrine formers, the fibrine in the shed blood amounting to 6, 8, or 10 parts per 1000 in place of 3 parts as is normal. The contraction of this causes a depression on the surface of the clot. 13. The red globules become viscous and adhere together by their flat surfaces to form rolls, which precipitate much more rapidly than single globules and leave the coagulated blood with a straw-colored upper stratum (buffy coat). 14. Increase of waste products, urea, uric acid, hippuric acid, etc.

The destruction and solution (hæmolysis) of blood-globules is greatly increased in certain inflammations and may be caused by a great variety of agents as taurocholic acid and taurocholates; blood serum from a different genus or species which has been injected with the blood of this species; venoms; toxins; ricin and other vegetable poisons, etc.

Other changes in the blood are alleged, like lessening of the albumen, as balancing the increase of fibrine, and lipæmia, but the constancy of these in all cases of inflammation is uncertain.

By way of comment and explanation of the above changes in the circulation the following may be advanced: The primary contraction of the capillaries is by no means a necessary condition of inflammation, and contractions and dilatations within certain limits occur in health and as a purely physiological act. The dilatation of the capillaries and the increased flow of blood to the part are related to each other as in part cause and effect, yet both are due to a reflex act from the seat of irritation which inhibits contraction in the capillaries and determines a more rigid contraction in the walls of the arteries running to the part. A rigid inelastic vessel of the same calibre and under the same pressure transmits more liquid than the one with elastic walls. The movement of the white globules to the walls of the vessel depends in part on their levity, light bodies passing into the outer slow moving layer, which is less dense, from the central stream where the force and density are greater. The epithelial cells of the intima

undergo cloudy swelling and are often detached, allowing the readier migration of the globules through the openings of the lymphatics and the softened and friable walls. When the capillaries are blocked the pressure necessarily increases on the arterial side, favoring laceration of the friable walls and the escape of minute masses of blood. The formation of the buffy coat is characteristic of the normal equine blood ; in inflammation it becomes more abundant. In the other genera a buffy coat apart from inflammation may be shown in : (a) anæmia or oligocythæmia in which the blood is deficient in red globules ; (b) in plethora in which there is an excess of blood solids ; (c) in pregnancy in which there is an excess of white and small red globules ; (d) in violent exertion or over-excitement, in which the blood has circulated with extraordinary rapidity. The all-sufficiency of the tissue cells in determining inflammation may be deduced from the following experiment. A ligature is tied around a frog's thigh so tightly as to arrest circulation, and the leg amputated above the ligature ; mustard is then applied to the web of the foot and a blister arises precisely as though circulation continued.

MICROBES, DIAPEDESIS AND PHAGOCYTOSIS.

The rôle of microbes in inflammation is much greater than was formerly supposed. It is now demonstrated that a large class of inflammations are directly caused by the colonization of microbes in the tissue and by the local irritation caused by their ptomaines and toxins. We must also admit the direct action of the latter on the heat-producing and vaso-motor nervous centres, as a factor more or less potent in different cases in the causation and maintenance of inflammation. No less important is the relation of the microbe to the migration of the globules and the subsequent results of the inflammation. This influence microbes share with certain chemical agents. Migration may be greatly checked even in inflamed parts by the hypodermic or intravenous injection of sulphate of quinia, eucalyptol, salicylic acid, or iodoform. Some have thought these acted by a chemiotactic attraction, but quinia is otherwise found to repel the leucocytes. Their action on the leucocytes or capillary walls is problematic.

Chemiotaxis is that power by which a microbe or any element

attracts or repels the leucocytes. When it attracts the chemiotaxis is said to be positive, when it repels it is negative. Among negative chemiotactic agents are quinia, solutions of sodium chloride (10%), and potassium salts, lactic acid, alcohol (10%), chloroform, glycerine, jequirity, and bile. To some agents (creatine, creatinine, allantoin, peptone, phlorydzine), leucocytes are indifferent. To gluten, wheat casein, pea legumin and the great majority of pathogenic microbes, leucocytes are positively attracted. As microbes exercise a great influence in producing local inflammation, so they are important factors in procuring an abundant emigration of leucocytes. Some of the most fatal microbes, like those of fowl cholera, repel leucocytes, and the benefit of their defensive work is to a large extent lost. The toxins of the chemiotactic microbe filtered from the bacteria exert the same influence as the living bacteria, as shown by Gabritchevski, Massart and Bordet.

But chemiotaxis may be exerted from within the bloodvessel as well as from without. Bouchard, Massart and Bordet have shown that a tube containing a culture of bacillus pyocyanus, introduced beneath the skin of a rabbit attracts in a few hours a great number of leucocytes. But if, immediately after its introduction ten cubic centimetres of a sterilized culture of the same bacillus are injected into a vein, very few leucocytes enter the tube inserted under the skin. The chemiotaxis seems to operate in this case from within the blood, and the desires of the leucocytes are satisfied without leaving the vessel. It would seem that in such cases the migration and protective work of the leucocytes are best exerted at the outset of the illness and before the toxic products have been poured into the blood in any quantity, whereas in the advanced stages when the blood is charged with ptomaines and toxins migration and phagocytosis would be likely to be limited and ineffective. The same consideration would forbid the use of drugs that check migration in all cases of attacks by microbes for which leucocytes have a positive chemiotaxis.

Phagocytosis is the act by which the leucocytes englobe and dissolve the invading microbe. By its amœboid movement the leucocyte flows around, and envelopes the microbe for which it has a positive chemiotaxis, and then begins the struggle of vitality between the two living germs. If the poison (leucomaine, anti-

toxin), and digestive ferment (enzyme) of the leucocytes are more deadly to the invading germ, than its ptomaines, toxins and enzymes are to the leucocyte, the white cell comes off the victor, and recovery takes place, but if the converse obtains the triumph is on the side of the microbe. As a rule much depends on the more or less deadly nature of the products of the invading microbe, on the numbers of the germ, the rapidity of its proliferation, and the consequent amount of its toxic products thrown into the system, on the one hand : And on the other on the potency of the chemiotaxis of the leucocyte for the invading germ, the number of white cells that emigrate into the inflamed tissue and engage in the work of phagocytosis, and on whether the particular animal system and its white cells have sustained a previous attack by the same germ and have thereby been educated to produce a greater amount of the defensive proteids (leucomaine, antitoxin, enzyme, etc.,) than they naturally would (acquired immunity).

Even with an abundant emigration of the leucocytes into the inflamed or invaded tissue, a number, greater or less, are usually destroyed by the microbial poisons and pass into degeneration or liquefaction, as in the formation of pus, and yet the attacking germ may be overcome, destroyed and devoured by the rapidly increasing survivors. In general terms the migration of the cells is in inverse ratio to the susceptibility of the animal to the microbe or the disease which it causes.

The positive and negative chemiotaxis, which determine phagocytosis or prevent it, may be seen in the action of the leucocytes toward the germs of two diseases, to one of which the animal is susceptible and to the other of which it is not. Thus the leucocytes of the pigeon take in the bacillus anthracis and suffer nothing apparently, whereas the same white cells of the dove are repelled by the bacteria of fowl cholera which are not therefore found in their interior.

The leucocytes that migrate from the bloodvessels are in the main, the most numerous, (the neutrophile or polynuclear) form ; the mononuclear leucocytes with horseshoe shaped nucleus also migrate but in much fewer numbers and are as a rule less occupied in phagocytosis. At the same time, these two forms may show each a preference for a particular microbe, the polynuclear

cell sometimes devouring one which the mononuclear cell rejects, and the mononuclear cell taking in one which the polynuclear refuses.

The small round white cells (lymphocytes) and the eosinophile leucocytes take no prominent part in phagocytosis.

EXUDATION.

In inflamed vascular tissues one of the most important results is the exudation. This is not, however, a mere transudation of the liquid parts of the blood, as takes place in dropsy, but it is to a large extent a selective process determined apparently by the condition of the capillary walls, and the nature of the inflammation is stated according to the character of the exudate. The dropsical effusion contains little albumen, fibrine or cell forms, and does not coagulate. The inflammation-exudate contains abundance of fibrine, cells and other solids and coagulates spontaneously in contact with inflamed tissue, or when removed from the body, by reason of the transforming leucocytes. Inflammatory exudate usually contains 6 to 8 per cent. of solids whereas the normal canine lymph contains 4 to 6. The exudate varies not only in different inflammations, but in successive stages of the same inflammation. The exudate may be mucous, serous, fibrinous or hæmorrhagic.

Mucous Exudate. In inflammation on a mucous or synovial surface the inflammatory exudation, mingled with the more or less altered secretion of the mucous glands, and the epithelial cells and leucocytes, forms a viscid fluid, rich in mucin, and characterizing the *mucous* or *catarrhal inflammation*. The nature of the discharge varies greatly, the serous character predominating at the start of the inflammation, and a thick, opaque creamy or semi-solid muco-purulent material appearing as the disease advances. It contains filaments of precipitated mucin insoluble in acetic acid or alcohol, and cells in all stages of change from the exudation leucocyte and mucous cell to the pus corpuscle, the latter being characterized by its bipartite or tripartite nucleus rendered visible by contact with weak acetic acid.

Serous Exudate. This consists of the liquid elements of the blood with only a limited amount of fibrine formers and conse-

quently little tendency to clot firmly. The presence of fibrinogen however, serves to distinguish it from the liquid of mechanical dropsy, as does also the greater quantity of cells and nuclei, of common salt and phosphates. It is usually straw colored in mass, but is sometimes slightly opalescent by reason of the numbers of cells and floating filaments of fibrine. Serous exudations take place in the early stages of inflammations (as in catarrh) and in inflammations of serous membranes (pleura, peritoneum, joints), in strong, vigorous subjects. They constitute the liquid contents of blisters whether raised by medicinal irritants, chafing, or heat. They clot under heat and nitric acid with a firmness proportionate to the amount of albumen.

These effusions are dangerous by reason of their interference with the functions of organs by pressure as with the dilatation of the lungs, the movements of the heart, the action of joints, or the integrity of the brain or spinal cord. When the causative disease has subsided they are usually speedily reabsorbed, the cells passing into the lymph vessels, or becoming degenerated, liquefied, and absorbed. Yet serous effusions often remain as permanent accumulations. For the blood staining of serous effusions and their clearing up, see under pleurisy.

Fibrinous Exudate. This is characterized by the amount of fibrinogen and fibro-plastin in its composition and by the comparative absence of leucocytes. It oozes through the vessels and coagulates in the tissues or on the surface of inflamed serous or mucous membranes. The more liquid part separating from the coagulum escapes from the free surface or accumulates in the lower part of the serous cavity. The coagulation is doubtless caused by the fibrine ferment derived from the rapidly proliferating cells and degenerating leucocytes. It usually occurs promptly in or on an inflamed tissue, but in contact with healthy structures only (as in a serous sac) it may remain fluid for an indefinite length of time. This exudate constitutes the false membranes that form on the pleura, pericardium or arachnoid, the coagulum of fibrinous pneumonia, and the plastic lymph on the surface of a granulating wound. It is especially injurious by reason of its enveloping organs (lungs, heart, bowels, iris) and subjecting them to permanent compression by reason of its contracting, also by binding them to adjacent structures by false

membranes. In coagulating it becomes first fibrillar then granular and finally undergoes molecular degeneration (Cornil and Ranvier), or development into new tissue (Paget). When organized it usually takes the form of the adjacent tissue from which its trophic cells are derived. Thus in divided tendons, in serous membranes and in granulating wounds it is fibrous, and between the ends of a broken bone it is osseous. If however, the adjoining tissue is a highly organized one, like nerve or muscle it may be replaced by a simpler (fibrous, osseous).

Fibrinous inflammations are especially found in connection with inflamed fibrous tissues and in strong vigorous subjects.

Blood Exudations. In all inflammations there is some migration of blood globules (red as well as white) but seldom in quantity sufficient to stain the tissues materially. Minute ruptures of the capillary vessels are not uncommon, with punctiform clots in the tissues, but extensive escape of blood is mainly seen in penetrating or contused wounds of the loose, subcutaneous connective tissue, and in infective inflammations (anthrax, Rinderpest, swine plague, petechial fever, malignant catarrh, snake-bites) with destruction of blood globules or extreme changes in the walls of the capillaries. Newly formed vessels in a friable neoplasm are subject to blood effusions. In acute inflammations of serous membranes the exudate is usually of a dark port wine hue at first. In such case it may pass in succession through all the stages of dark red, brick red, yellow, reddish, and chocolate color, before becoming milky and finally transparent.

Croupous Exudate. Croupous inflammation usually occurs on or near a mucous surface and is characterized by an exudation consisting mainly of fibrinous material entangling white cells, epithelium, a few pus corpuscles and some form of bacteria. In true diphtheria of children this is the Löffler bacillus, in the pseudodiphtheria, attending on scarlatina, etc., it is often streptococcus pyogenes, and in the diphtheria of calves it is bacillus diphtheriæ vitulorum. Pseudomembranous inflammations therefore constitute a group agreeing in the nature of the exudate but differing essentially in the cause. This difference in the cause has a most material effect on the course and gravity of the disease. One form like true diphtheria in man not only extends into the tissues, and tends to necrotic changes, but also poisons the nerve

centres by the toxic materials absorbed inducing troublesome paralysis, while another like croup of children establishes a violent but essentially superficial disease and when that recovers it leaves no ulterior ill effects elsewhere.

A **Chyliform exudate** has been noted in peritonitis in the dog, the milky whiteness being due to fatty granules.

RESULTS AND PRODUCTS OF INFLAMMATION.

As nearly all inflammations have significant exudations it is well to follow these in their subsequent progress through reabsorption and removal, development into new tissues, necrosis, suppuration or ulceration.

Resolution. If an inflammation, slight in character and with only a moderate exudation, subsides and is followed by a rapid liquefaction of the cells and fibrinous coagula and a reabsorption of the exudate, so as to leave the part in its primary healthy condition structurally and functionally, it is said to have terminated by "*resolution*." If this occurs with extraordinary rapidity it is said to have ended by "*delitescence*." This is not always an unalloyed good, as often in delitescence, coagula and infecting material may be carried on by the circulation, to block the next set of capillaries in its course and set up new centres of inflammation. This is one form of "*metastasis*" though a more definite *metastasis* is in rheumatism, where the disease attacks one joint to-day and a distant one to-morrow.

Inflammatory New Formations. Of the growths in lymph there are two principal kinds: First, *the plastic, fibrinous, granular or molecular*; and second *the aplastic or corpuscular*. The first form tends to develop into new structure, the second to disintegrate and decay. The tendency to one or other form depends largely on the strength or weakness of the system's health, on the deficiency or excess of corpuscles in the exuded fluid, and on the distance of the latter from living tissues and blood supply. Much also depends on the predisposition of the genus, the tendency to suppuration in lymph being in a descending series from horse, ass, and mule, through ox and sheep, to dog, pig, and finally, the bird, in which latter suppuration is quite exceptional.

Suppuration. In inflammation of a high type, in those oc-

curring on the skin or mucous membranes in which there is an extraordinary increase of nuclei and embryonal cells, and in lymph thrown out in excess at one point, so that its central parts are far from vascular tissue and nourishment, the cell elements undergo a rapid increase and degradation into pus-corpuscles, and its solidified intercellular lymph undergoes granular decay and liquefaction into pus.

While the above conditions are favorable to the formation of pus, the process of suppuration must now be recognized as an infective process due to the propagation of bacteria (mainly chain forms—*Streptococcus pyogenes*—cluster groups—*Staphylococcus pyogenes*—and rod forms—*Bacillus pyogenes*). These or other bacteria are found in the pus of acute abscesses, and when absent in chronic abscesses are to be considered as having perished since the abscess was recent and active. Inoculation of a rabbit with an excess of the pus of an acute abscess produces general purulent infection (pyæmia) and early death; from a medium dose an abscess is produced; while from a small dose there is no effect whatever. In the latter case the bacteria are overcome and devoured by the abundance of vitally potent white blood-globules and tissue cells. This pus-forming action of these bacteria explains the great difference in results in wounds exposed to the air and those in the interior of the body and far removed from air and its floating bacteria. A broken bone, with no wound in the skin and little injury to parts around the fracture, is readily repaired without any formation of pus, if merely kept still and immovable; whereas a broken bone, continuous with a wound through the skin, always tends to form pus or become otherwise infected, and is extremely dangerous even to life. The tendency of every open sore is to form pus on its surface, but this may be arrested and avoided by preventing the access of germs, as by a free use of disinfectants or a covering which shall arrest and filter out the germs. Similarly in an abscess, evacuation followed by the injection of disinfectants, without the formation of any perceptible permanent opening to the outer air, will put a stop to the pus-formation. The subjection of an inflamed part to the control of these pus-forming bacteria is dependent on the lowered vitality and power of resistance of the inflamed tissues, and of the white cells of their circulating blood. Healthy parts can success-

fully resist them, though they are constantly present in surrounding air and on objects, but in this as in all other cases of bacterial infection, so soon as the tissue is injured, inflamed and lowered in its power of vital resistance, the pyogenic bacteria assail it successfully. Hence, too, the more abundant exudations of lymph, the centres of which are farthest removed from the healthy, resistant tissues and from nourishment, are the most prone to suppuration. That the germs can make their way to such deep-seated exudations in the substance of solid tissues is to be accounted for by their gradual advance through the inflamed and weakened structures from the adjacent skin or mucous membrane, or in some instances by reason of their presence in small numbers in the blood. It is further noteworthy that those animals in which suppuration does not occur readily are such as have a special power of resistance to some other organic poisons. Thus the hog, which is supposed to be proof against snake-bite, is also, to a large extent, proof against the pus-forming bacteria.

Pus. This is a white, or yellowish-white, creamy-looking product, composed of a clear, transparent fluid, rendered opaque by numerous floating pus-corpuscles. These pus-corpuscles have the same size as the white globules of the blood ($\frac{1}{2500}$ to $\frac{1}{3000}$ inch) and are peculiar in that each shows within it three or more nuclei, which become visible on the addition of a drop of water or acetic acid. Each of the common embryonal cells found in the inflamed tissue usually contains two nuclei, the indication of the active increase by division into two, but when the supply of nutriment is checked the nuclei continue to divide, while the cells remain unchanged, and thus every cell comes to contain several nuclei in addition to fatty granules, and constitute pus-corpuscles.

When pus is formed in a well-maintained system and tissue, the outer layer of the lymph is developed into a fibrous sac inclosing the liquid pus and constituting an *abscess*. In an unhealthy system, or when the inflammation depends on some injurious poison, like that of erysipelas, this sac may not be formed, and the pus, burrowing into and between different organs, destroys the connections and substance—*diffuse suppuration*. When an abscess has formed in soft tissues its investing sac shrinks as it assumes the fibrous character, and the confined

pus being incapable of compression, presses the membrane outward on the side in which the surrounding tissues are most loose and least resistant, hence, usually, though not always, in the direction of the skin; the soft tissues become absorbed and removed in the track of the advancing pus; and, finally, the latter reaches a free surface and escapes. Thus, an abscess usually bursts through the skin, but also, at times, through a mucous membrane into the lungs, bowels, etc., or through a serous membrane into chest, abdomen, etc. When an abscess is formed in bone or dense fibrous tissues which press equally on all sides, it may remain imprisoned for months and years after all inflammation has subsided, constituting an *indolent* or *cold abscess*. When the imprisoned pus is inclosed by thick fibrous or resistant tissues at all points but one, it will make its way along the narrow passage of yielding tissue, but as the resulting outlet is constricted, long, and tortuous, the contents cannot readily escape through it nor the walls of the abscess contract so as to expel the confined pus, and the latter goes on forming and discharging through the narrow outlet for months or years. This is a *fistula* or *sinus*.

Healing by Adhesion or First Intention. When a clean-cut wound has the blood staunched and its lips brought together without exposure to the air (or contact with pyogenic germs), they adhere at once and heal without pus or almost any appreciable formation of new tissue. Here the lymph thrown out on the cut surfaces agglutinates them, and the cells, multiplying, form a thin layer of embryonic tissue which gradually develops into a fibrous structure and repairs the breach without any perceptible scar.

Healing by Second Intention. Granulation. When a wound has caused destruction of tissue, or when a simple incision is left exposed to the air, the breach is filled up by new tissue through the process known as granulation. The superficial layer of lymph thrown out on the raw surface becomes oxidized and degenerates into pus, while the deeper layers become solid, fibrillated, the seat of cell-growth, and are finally transformed into a fibrous structure. New blood-vessels form in loops in the developing lymph and constitute the bright-red granulation-points which cover the raw surface. The fibrous tissue into which the lymph is transformed undergoes gradual contraction in develop-

ment, and thus, day by day, the edges of the adjacent healthy skin are drawn in, so as to cover the wound more or less perfectly, and a slight scar only is left when healing has been accomplished.

Granule Corpuscles and Masses. This is another degenerative transformation in lymph and, is seen mainly in inflamed glands and brain and lung-tissue. The cells found in the exuded lymph are made up of minute granules, anabolic, catabolic, etc., and besides these, large, irregularly shaped masses of granules are extended along the capillary blood-vessels. After the lymph has coagulated these granular masses soften and liquefy preliminary to re-absorption and removal, and the restoration of the tissue to a healthy condition. When in excess this softens and disintegrates the tissues, leading to permanent loss of substance. See *granular* degeneration.

Interstitial Development of Lymph into Tissue. This is equivalent to what takes place in the formation of the sac of the abscess or of granulation-tissue. The liquid lymph in coagulating, becomes fibrillar, and the cells and nuclei of the adjacent tissue, having an abundant supply of blood and nutriment, multiply first as simple, rounded, embryonic cells, then deposit around them new tissue, becoming elongated, spindle-shaped, branching, etc., and thus get imbedded in a fibrous material of their own formation. These new formations are usually of a low type of organization, like white fibrous tissue or bone, and hence, although breaches in the higher structures like muscle, nerve, gland, skin, are filled up, it is usually only by the drawing together of the remaining healthy parts by these new formations without the restoration of any of the original tissue which has been destroyed. The cicatrix (scar), alone is made up of new material.

Lymph developing in this way may undergo any degeneration to which normal tissues are subject. Thus it may undergo black, pigmentary (*melanotic*) degeneration, it may become impregnated with lime-salts (*calcified*), it may wither up into a hard *gelatini-form* or *horny* mass, or it may undergo *fatty* degeneration.

Fatty degeneration is the most common form, and consists in the excessive deposit of fatty granules, first in the cells which are in excess or badly nourished, and next in the adjacent tissue, the normal elements of which are replaced by fatty granules.

Softening is an almost constant result of inflammation. The

exudate infiltrates and separates the tissue elements, destroying their cohesion; the liquefaction of these elements impairs this still further, and the more or less perfect transformation of the tissue into embryonic tissue entails the loss of its rigidity and power of resistance. Thus the inflamed brain-tissue may become a mere pulp, and the inflamed bone may be cut with a knife.

Ulceration is closely allied to softening. On the surface of a sore there is an excessive exudation of lymph which loosens and disintegrates the layer of lymph that is already in process of development, and also a part of the tissue beneath. The cells in these parts fail to develop naturally and to build up good tissue; they become fatty, die, and together with the tissue in which they lie, break down and pass off as a pulpy débris. Thus the sore constantly deepens and widens, or at least refuses to contract and heal. It is usually the result of bacterial infection.

Gangrene or death of a part is another effect of inflammation. It results usually from the cutting off of the blood-supply through the obstruction of the blood-vessels; by the pressure of excessive exudation in unyielding structures, as in bone, or under the hoof; by implication of the inner coats of the blood-vessels in the inflammation, when the contained blood will clot and obstruct them; or by blocking with the blood-clots that have been formed at a distance and washed on in the blood-current to be arrested when they reach vessels too small to admit them. Like suppuration, gangrene is associated with and often caused by a bacterial growth. The dead mass remains as an irritant, and is slowly separated by the formation around it of embryonal tissue, granulations and pus. A second form is *molecular gangrene*, in which the cells and minute elements of the tissue die, and are cast off, leading to phagedenic (eating, extending) sores, as noted above under Ulceration. When gangrene occurs on an exposed surface, that may be altered from the normal color into shades of yellow, brown, green, red, or black, according to the amount of blood and the stage of decomposition, and may be cut without pain, if the subjacent parts are not pressed upon; it may be soft, may pit on pressure, may crackle under the hand from the evolved gases of decomposition, and may be covered with blisters (*phlyctenæ*) with red, grumous liquid contents (*moist gangrene*); again, it may be white, as after freezing, or it may be dark-colored, dry, and horny, as from ergotism (*dry gangrene*).

FEVER.

Definition. Symptomatic. Idiopathic. Symptoms. Contagion. Incubation. Premonitory symptoms. Chill, rigor. Reaction, hot stage. Defervescence. Crisis. Lysis. Natural temperature. Fever temperature. Retention of water in the system. Production of waste materials. Typhoid condition. High fever, low, hectic. Treatment in vigorous subject, in weak one. Regimen. Solipeds. Ruminants. Carnivora. Drink. Rest. Clothing. Air. General and local bleeding. Cupping. Warm baths, tepid, compresses, derivatives. Cold. Diaphoretics. Laxatives, Diuretics. Sedatives. Alkalies. Antipyretics. Stimulants. Tonic refrigerants. Tonics. In low fever. No depletion. Judicious elimination. Stimulants. Refrigerants. Antiseptics. Diet. Local treatment of inflammation. Cold. Astringents. Antiseptics. Warm applications. Stimulating embrocations. Blisters. Firing. Massage. Suppuration.

Definition. Whether occurring as an accompaniment of inflammation or independently of it, fever is an unnatural elevation of the temperature of the body, the direct result of an excess of destructive chemical change in the blood and tissues, and more remotely of disordered nervous function.

Of all extensive inflammations fever is the constant result and accompaniment, rising as the inflammation rises or extends, and subsiding as the inflammation subsides. It also occurs as a distinct affection, as in all the infectious diseases, as the result of a specific irritating poison in the system, and then is the manifestation of the disease, while a local inflammation may or may not be present as a special secondary feature of the malady or as an accidental complication.

Symptoms of Fever. Fever is marked by certain definite stages, each of which has its own special manifestations. In the cases due to a specific disease-germ, or *contagium*, these are, however, preceded by a period of *latency* or *incubation* in which no symptoms whatever are manifest, but during this time the germ is rapidly multiplying in the system, and it is only when it has gained a certain increase that it disorders the nervous system, wastes the tissues, raises the temperature of the body, and induces the other phenomena of fever. The same may be said to hold in the fever attending on inflammation. The slight and circumscribed inflammation is at first productive of no fever, and it is

only when it gains a certain extent that the nerves and nutrition are disordered so as to bring about a feverish condition.

Premonitory Symptoms. These usually last but a few hours and are often entirely absent or unnoticed. There is a lack of the customary vigor and spirit, an indisposition to exertion, a loss of clearness and vivacity of the eye, a manifest dullness, with hanging of the head, and frequent shifting of the limbs as if fatigued. Appetite is less sharp and ruminants chew the cud less heartily or persistently.

Cold Stage. These are soon succeeded by the *chill*, *rigor*, or *shivering fit*, in which the hair, especially that along the back, stands erect (staring coat), the skin is cold and adherent to the structures beneath (hidebound), the extremities (legs, tail, ears, horns, nose) are cold, and the frame is agitated with slight tremors, or even a shivering so violent that a wooden floor or building is made to rattle. The back is arched, the legs brought nearer together (crouching), the mouth is cool and clammy, the breathing hurried, the pulse weak, and it may be rapid, but with a hard beat, the bowels costive, and the urine higher colored than natural. The temperature of the interior of the body, taken by a thermometer in the rectum, is already found above the normal, the excessive destruction of tissue having begun, and the blood driven from the cooler surface, and accumulating in the hot interior, at once favors tissue-change and maintains the extra heat thereby produced. In cattle the end of the tail is soft and flaccid from this stage onward. The *cold stage* lasts a few minutes, or one or two days in different cases.

Hot Stage. The hot stage appears as a reaction from the chill, the contraction in the minute vessels of the skin giving place to dilatation, so that the whole surface, including the extremities, becomes hot and burning, but still dry and parched. The burning is especially noticeable in the more vascular parts, like the roots of the horns and ears, the muzzle or snout, the mouth, the hoofs, the bare parts of the paws in carnivora, and the mammæ (udder) in milch animals. The mucous membranes lining the nose and mouth become hot and red, the breathing freer, but not less rapid, the pulse softer but accelerated, appetite (and rumination) greatly impaired or lost, thirst great, costiveness increased, urine diminished and of a higher color, the flow of milk greatly

impaired or entirely arrested, and the dullness and prostration greatly increased.

The hot stage lasts longer than the cold one, usually persisting until death or convalescence. It may alternate with chills throughout the whole course of the illness, and in the fever of inflammation the interruption of the *hot stage* by a chill usually implies either a considerable extension of the inflammation or the occurrence of suppuration.

Defervescence. The decline of the fever may take place by a sudden reduction of the body temperature to the natural standard, or near it, and a sudden and general improvement in the symptoms (crisis), or by a slow improvement from day to day through a more or less tedious convalescence (lysis).

Fever Temperature. A temporary rise of one or two degrees is unimportant, but a permanent rise indicates fever. A rise of ten or twelve degrees is usually fatal. A sudden fall to or below the natural, unless with general improvement in the symptoms indicates *sinking*. A similar fall, with a free secretion (perspiration, urination, relaxed bowels) and general improvement in symptoms, betokens recovery. For normal and febrile temperature see Semeiology.

Retention of water in the fevered system is as significant as the elevated temperature. The patient drinks greedily but all the secretions are arrested or diminished, and liquids go on accumulating in the system. The sudden bursting forth of secretion (especially sweating) implies that the fever has, at least temporarily, given way.

The production of waste matters in the system is necessarily proportionate to the amount of tissue destroyed. This appears in the blood mainly as urea, the organic acid of urine (uric mainly, hippuric in sound herbivora), together with phosphates, sulphates, and chlorides. These thrown off by the urine give it its high density. If not thus thrown off they remain as poisons in the circulation and bring about that prostrate, sunken, debilitated condition which characterizes the advanced stages of all severe and continued fevers—the *typhoid condition*. This is not to be confounded with the specific *typhoid fever*, in which a special fever germ expends itself, mainly on the bowels, and that runs through a regular course. The *typhoid condition* is that state in

which an animal system, already greatly weakened by a severe disease, and perhaps further prostrated by a specific disease-poison, is subjected to a species of poisoning by the retained chemical products of the waste of the tissues.

Types of Fever. These are as characteristic as the types of inflammation, and of the same kind. The *strong* type of fever which attends on an acute inflammation in an otherwise healthy vigorous system, is spoken of as a *high* or *inflammatory* fever. The *weak* type which occurs in a broken down or debilitated system, or in connection with the action of a specific disease germ, or with the saturation of the system by waste chemical products is known as *low*, *typhoid* (better *typhous*), or *adynamic* fever. That form which persists in the utterly debilitated system, where the power of assimilation is practically lost, is known as *hectic*.

TREATMENT OF INFLAMMATION AND FEVER.

Treatment will be guided very largely by the type of the attendant fever. If that is of a high type, with a hard, full, rapid pulse, bright red mucous membranes, a clear eye, and well sustained strength in a strong, vigorous animal, what is known as antiphlogistic (depleting, depressing) treatment is admissible at the outset. But in many cases with a low type of fever, a weak, rapid pulse, pallid, yellow, or livid mucous membranes, a coated tongue, a dull or sunken eye, much depression and prostration, swaying on the limbs in walking, pendant head, ears, eyelids and lips, and varying and irregular temperature of the limbs, etc., such measures are forbidden from the first, and tonics and stimulants are demanded from the outset. Between the two extremes there are many grades, which demand a judiciously adjusted intermediate treatment. The general principles only of each characteristic form of treatment can be here formulated, it being understood that no two cases can be most advantageously treated in precisely the same way, but that according to its special grade each case will demand its own specific management applied according to the skill of the physician.

Regimen. An antiphlogistic diet will consist in a moderate or very sparing amount of non-stimulating food of easy digestion (wheat bran or oil meal in warm, sloppy mash, carrots, turnips,

beets, potatoes, apples, pumpkins ; fresh, tender, green grass or in winter a little scalded hay, may be taken as examples). *Ruminants* should have no food necessitating chewing of the cud ; thus the roots, etc., should be pulped or boiled, and hay and even grass must be interdicted until rumination is re-established. When food is absolutely refused for days in succession well-boiled gruels of oat-meal, barley-meal, linseed-meal, bran, etc., may be given from a bottle or by injection. *Dogs* and *cats* should have only vegetable mush (unbolted flour, barley, or oat-meal) with just enough beef-juice to tempt the animal to eat a little. Milk with an admixture of oxide of magnesia, or even lime-water is often at once palatable and cooling. *Drink* should be pure water, cool, if kept constantly fresh before the animal, but warmed to something less than tepid if supplied only at long intervals, so that the thirsty patient is not tempted to drink to excess and chill himself. *Rest* in a clean, well-aired building, free from draughts of cold air and with a southern exposure, is desirable, especially in winter. The best temperature is usually sixty degrees to seventy degrees, especially in inflammations in the chest, and extremes of temperature are to be avoided. *Clothing* will depend on the weather. In warm weather it may be often discarded, while in winter it should always be sufficient to obviate the access of *chill* and consequent aggravation of the disease. Whenever the atmosphere can be kept warm only at the expense of impurity it is better to secure the comfort of the patient by the requisite clothing than to subject him to impure air. As the extremities are the first to suffer from cold, loose flannel bandages to the limbs are often imperative.

Remedies. *General bleeding*, a great resort of our fore-fathers, has been long all but discarded from modern practice. To-day it is rarely resorted to, except to save from an urgent and extreme danger, as in the plethoric cow merging into parturient apoplexy, or the fat and overdriven horse, gasping for breath and life, in general acute congestion of the lungs. There are other cases of extensive acute and dangerous congestions, especially in a strong, vigorous, and plethoric patient, in which general bleeding is beneficial in warding off threatened death ; but sound, discriminating judgment is necessary to its safe employment. When resorted to at all, the blood should be drawn from a large

orifice, in a full stream, to secure the desired depressant effect with the smallest loss of blood, and the patient should be kept especially quiet and apart from all excitement which would tend to counteract the sedative action. The flow should be stopped as soon as the pulse softens.

Local bleeding is more extensively applicable than *general*, as it usually effects the same purpose without the permanently weakening effect. It acts in two ways, first, by emptying and contracting the vessels in the skin over the inflamed organ, it solicits a sympathetic contraction of the capillary vessels in that organ itself, and thus inaugurates a progress toward recovery; and second, by so much as it draws blood to the surface it diminishes the blood-pressure on the deeper inflamed organ, and affords a better opportunity for the restoration of the healthy circulation and function. Local bleeding may be practiced by simple scarification or leeches, or better, by cupping with or without scarification. To apply leeches, the skin must first be shaved. To cup, it must at least be greased. As a cup, an ordinary large drinking-glass may be used, the air contained in it being driven out by a lighted taper, and then the taper being withdrawn, the mouth of the cup is instantly and accurately applied on the skin and held there, until, as it cools, it draws up the skin within it and clings like a sucker. A number of these may be applied according to the extent of the inflammation, and, if desired, they may be removed, the part scarified, and the cups reapplied. The cupping usually effects more than a mere local attraction of blood; it very commonly causes a free circulation in the whole skin, a generally diffused warmth, and even perspiration. Thus we may secure the derivation of blood from the inflamed part, the cooling of a large mass of blood in the extensive cutaneous circulation, the cooling of the entire system by the return of this blood internally, the elimination of injurious waste matters through the skin, the lowering of the febrile heat and tension, and a better functional activity of all the organs of the body.

Similar good results are obtained from all remedies that induce surface warmth and vascularity and a free secretion from the skin.

Warm baths, for animals to which they can be applied, abstract blood temporarily from the inflamed internal organs, diminish the

blood-pressure, and really cool the system, beside securing elimination from the skin and other secreting surfaces. They may be commenced warm (80° F.) and gradually cooled down to 65° F. after the skin has become freely active. In the larger quadrupeds, in which the warm bath is too often practically impossible, the same revulsion of blood and warmth to the skin may be secured by *rugs wrung out of hot (almost scalding) water*, wrapped tightly round the body, covered with two or more dry blankets, and kept tightly applied against the surface by elastic surcingles. The legs may be rubbed with straw wisps till warm, and then loosely bandaged, or applications of red pepper, ammonia, or mustard, may be made prior to bandaging. In place of hot water rugs, bags loosely filled with bran, chaff, or other light agent, heated to 110° F., may be applied round the body, or, where it is available, a Turkish or steam bath may be resorted to. These hot cutaneous applications, to produce glow and perspiration, are especially valuable in the chill that heralds a violent inflammation, and if that can be suddenly checked by this means the inflammation will often be warded off, or at least rendered slight and easily controllable. After perspiring for half an hour the patient may be gradually uncovered, rubbed dry, and covered with a dry, warm blanket. If the skin is still glowing, a slight sponging with cool or cold water may beneficially precede the rubbing and drying.

Cold Baths. In cases of very high fever a full cold bath (68° F.) may be employed for fifteen minutes, and repeated as often as the temperature rises. In many cases of parturition fever in cows great benefit accrues from sponging the body with cold water and allowing it to evaporate from the burning skin. In the extreme fever of heat apoplexy (sunstroke), with a temperature of 110° F. and upward, a strong current of cold water from a hose directed on the head and body often gives the best results. In ordinary fevers in large animals the *cold pack* will often serve a good purpose. Wring a blanket out of water (cold or tepid, according to the height of the fever and the strength and power of reaction of the patient), wrap it round the body, cover it with several dry blankets so that no part is exposed, and keep the whole in close contact with the skin by elastic surcingles. In fifteen minutes the skin should be glowing and perspiring, and

in half an hour the wrappings should be removed, a little at a time, the parts rubbed dry and covered with a dry woollen blanket. It may be repeated as often as the fever rises.

Diaphoretics. Besides these remedial methods of inducing a revulsion and glow in the skin with perspiration, *medicinal diaphoretics* may be resorted to. Among these may be included *copious drinks and injections of warm water, acetate of ammonia, antimony, ipecacuan, or pilocarpin*, or one of the sedatives, *aconite veratum, or opium*, etc. Many a threatened acute inflammation has been to a great extent cut short and nipped in the bud—the stage of chill—by warm clothing, active hand-rubbing, and such an apparently unscientific nauseant as tobacco.

When the preliminary stage has passed and the hot stage of the fever has set in, cooling and eliminating agents are especially called for.

Laxatives. In many cases, and especially in those with marked constipation or bowels loaded with indigestible materials, a laxative is beneficial. For the horse, aloes, or, often better, sulphate of soda, and for cattle or sheep, the latter, or Epsom salts, will at once remove an irritant, cool the general system, draw off much blood and nervous energy to the bowels, and secure a considerable depletion and elimination from the intestines. For swine, dogs and cats castor-oil or salts may be used, and for fowls castor-oil. Arecolin or phenolphthalein may be used. If the mucous membranes are yellow, the tongue furred, and fæces scanty, hard and foetid, a dose of calomel (horse or ox, one drachm; sheep or pig, one scruple; dog, three grains; chicken, one-half grain) with tartar emetic (horse or ox, two drachms; sheep, twenty grains; swine, one-half grain; dog, one-fourth grain; chicken, one-eighth grain) may be given and followed in ten hours by one of the laxatives named above.

Diuretics. In the absence of any manifest disorder of the digestive organs, the laxative may be omitted and refrigerant diuretics resorted to. Acetate of ammonia or potassa, nitre, tartrate of potassa, carbonates of potassa or soda, may be used along with sedatives.

In cases of infectious disease with poisoning by ptomaines and toxins the elimination of these by the bowels and kidneys is of the greatest importance.

Sedatives. Of the sedatives, aconite, bromide of potasium, veratrum, hyoscyamus, or chloral hydrate may be used according to the special indications.

Alkalies. Resolvents. When the organ inflamed is a serous membrane in which dangerous adhesions or other functional disorders are likely to occur from newly formed false membranes, their formation should be counteracted as far as possible by the free use of alkalies (carbonates of soda, potash, or ammonia, nitre, iodide of potassium, muriate of ammonia. etc.), and in the same conditions excessive effusion should be controlled by free action on the kidneys.

Antipyretics. To reduce the febrile temperature and especially, when caused by the ptomaines and toxins of bacterial infection, agents like acetanilid, antipyrin, exalgin, creolin, benzanilid, salicylate of soda, and quinine have been largely employed and will usually lower the temperature several degrees in a few hours. They nearly all depress the vital forces, or hinder reparatory processes, so that their use is to be carefully guarded. Quinine which is less depressing than the others hinders migration of the leucocytes and thus stands in the way of successful phagocytosis. With a dangerously high temperature they may be temporarily admissible, but they should be suspended as soon as possible. In all ordinary cases they are probably better avoided. A judicious use of the cold or tepid bath, or of wet compresses is incomparably safer and more generally applicable.

Stimulants. When the disease results in great prostration or when symptoms of septic or ptomaine poisoning set in, stimulants are often required to sustain the flagging heart and circulation. These may be alcohol, ammonia, ether, camphor, digitalis, etc.

Tonic Refrigerants. Later, when both inflammation and fever have been somewhat reduced, temperature, breathing, and pulse rendered more moderate, eye clearer, and even appetite perhaps slightly improved, the sedatives may give place to refrigerating tonics, such as mineral acids (nitric, muriatic, sulphuric, or phosphoric), in combination with bitters (quassia, cascarilla, calumba, gentian, salicin), without as yet the suspension of refrigerant diuretics. Thus for the horse the following : Recipe : Pharmaceutical nitric acid, two drams ; infusion of gentian, ten ounces ; nitrate of potassa, two ounces. Dissolve. Give one ounce every six hours.

In Convalescence. When convalescence has fairly set in, the fever has subsided, and there remains merely some debility with a remnant of the inflammatory exudation to be removed or organized into tissue, or when an abscess has developed and burst, the tonics must be more freely given, the mineral acids may even give place to preparations of iron or cod-liver oil, and the diet must be made increasingly liberal. But throughout the whole progress of the disease the bowels should be carefully watched. Costiveness may quickly undo all that has been gained, hence any indication of this should be met by laxative food (boiled flaxseed, etc.), or, this failing, by injections or laxatives. Similarly, if a freer action of the kidneys seems to be necessary for elimination of waste matters or to reduce fever, diuretics should be continuously kept up.

TREATMENT OF ADYNAMIC INFLAMMATION AND FEVER. In treating *low asthenic* or *adynamic inflammation* all depression and depletion is to be carefully avoided. Even *laxatives* must be employed with extreme caution. If absolutely necessary it is best to give them in small (half) doses and supplement their action by liberal injections of hot water. Elimination of waste matter from the blood and system is still to be sought, but it must be by *stimulating diuretics* (sweet spirits of nitre, carbonate, acetate, or muriate of ammonia, digitalis), and direct *stimulants* and *tonics* must be given from the first (ammonia, wine, strong ale, whisky; brandy, ether, gentian, calumba, nux vomica). For the horse the following may serve as an example: Recipe: Sweet spirits of nitre, four ounces; sulphuric ether, two ounces; tincture of gentian, ten ounces; digitalis, one dram. Mix. Dose, two ounces in a pint of cool water four times a day. When there is great debility and prostration ammoniacal and alcoholic stimulants must be given freely, while if the fever heat rises very unduly the cooling diuretics (citrate, tartrate, or acetate of potassa, or nitre, etc.), and even sedatives (bromide of potassium, hydrobromic acid, chloral hydrate, salicin, salicylate of soda), must be resorted to. If there is any indication of a special depressing poison in the system, or of the absorption of septic or other noxious matter from a wound, antiseptics (hydrochloric acid, or salicylic acid, sulphite of soda, quinia, or chlorate of potassa) may be advantageously added to the prescription.

In these cases of asthenic inflammation, as in the advanced and debilitating stages of sthenic inflammation, the diet should be as good as the patient can digest. Boiled oats, barley, or flaxseed, rich, well-boiled gruels, and beef-tea (even for herbivora,) may frequently be resorted to with advantage.

Local Treatment of Inflammation. In all forms of superficial inflammation the local treatment occupies an important place. The persistent application of *cold* (cold water in a stream, ice-bags, freezing mixtures) will sometimes overcome the tendency to inflammation or arrest it. This is especially sought when a violent inflammation (as in a wounded joint) threatens to destroy an important organ. If adopted, it must be persisted in, as if it is suspended too soon the reaction is likely to make matters worse than ever. *Cold astringent applications* have a similar tendency. Sugar-of-lead, one-half ounce ; laudanum, one ounce ; water, one quart, may be kept applied by means of a linen bandage. The water may often be advantageously replaced by extract of witch-hazel. If the inflamed part is superficial the lotion may be made *antiseptic* (carbolic acid, one dram ; or sulphurous acid solution, five ounces ; water, one quart). *Hot applications, fomentations, poultices* are nearly always appropriate but they should be made antiseptic to prevent bacterial development. When adopted they should like *cold* ones be kept up as continuously as possible. These soothe alike the superficial and deeper parts, the latter through sympathy, producing first a relaxation of vessels and tissues, and later a contraction of the former attended by pallor of the surface. They greatly favor suppuration when that is already inevitable, though in other cases they may obviate it by checking at an early stage the acute inflammatory process on which it depends. Any bland agent that will retain heat and moisture will make an excellent poultice, though flaxseed-meal is the type of a soothing demulcent application. Very slight inflammation may be successfully treated at the outset with a *stimulating embrocation* (alcohol or camphorated spirit), yet in the more violent type of acute inflammation all local excitants tend to aggravate the disease. In these violent forms the activity of the disease should be first abated by local soothing and general sedative measures, and then the part over the inflamed organ may be safely treated with a stimulating liniment or even a blister. In such

cases the liniment first acts as a derivative of blood and nervous energy from the inflamed part, and later and still more beneficially by securing in it a sympathetic healing process, like that set up in the skin. It is further probable that the absorbed albuminoids, which have been modified in the congested part often exercise a decided effect on the inflamed tissue. In raw sores where inflammation has been set up, the granulations may become dropsical or excessive, bulging beyond the adjacent skin as *proud flesh*. This should be repressed by touching it gently with some mild caustic (lunar caustic), so as to produce a thin, white film, and the remote cause of the inflammation (often a local irritant) should be sought and removed. In some unhealthy sores tending to excessive granulation, the compound tincture of myrrh and aloes may be applied daily with great benefit. When the granulations become excessive they may be scraped down to the level of the skin and then treated with an antiseptic (iodoform, boric acid, acetanilid, aristol.)

Blistering. In subacute and chronic inflammations and in those acute forms in which the violence of the inflammatory action has been already subdued by soothing measures, blisters and other counter-irritants may be employed to counteract the remaining inflammatory action. These act primarily by drawing off blood and nervous energy from the inflamed organ to the skin, and secondarily, by establishing a sympathetic healing process in the diseased part, simultaneously with the work of recovery in the skin, when the blister has spent its action. But if applied above a part which is still violently inflamed, there is apt to be serious aggravation, through this same sympathy with the part suffering under the rising of the blister. In this way great and irreparable injury is often done through the laudations of particular blisters for the cure of given diseases, without any reference to the stage or grade of such disease. The value of a blister depends far more on the time of its application than on the ingredients of which it may be composed.

Firing. This acts in nearly the same manner as a blister, and demands similar caution in its application. It is especially available in subacute and chronic diseases of the joints, bones, and tendons, and may be made more or less severe according to the nature and obstinacy of the disease. It is applied in points or in

lines at intervals of one-half to one inch, and penetrating one-third, one-half, or entirely through the skin. The hotter the iron the less the pain, but the greater the danger of destruction of the intervening skin by the excess of radiating heat. Hence the contact of the heated iron with any one part must be judiciously graduated to the heat of the iron and the delicacy of the skin, and should not exceed the fraction of a second.

Massage, Rubbing. In chronic inflammation and even in some acute forms, with considerable exudation, rubbing or massage is of great value. It hastens the progress of the blood through the veins, tends to restore the normal circulation in the stagnant or partially obstructed capillaries, moves on the exuded liquids in the lymphatic plexus, rendering the absorption more active, and at once prevents the process of disintegration of the tissues and obviates the necessity for their solution and removal. This may be largely accomplished by the use of the brush or rubber, or by careful manipulation especially in the direction of the veins. If the inflammation is near the surface the use of antiseptic and deobstruent agents will heighten the good effect. Iodoform, iodide of potassium, boric acid may serve as examples.

Suppuration. Abscess. The great variety of the causes and forms of suppuration would forbid any extended notice of its treatment in this place. It seems preferable to refer the reader to the subject of pyæmia and the various surgical and medical diseases in which suppuration takes place.

DISEASES OF THE RESPIRATORY ORGANS.

Importance of diseases of the respiratory organs—in horses and dogs. Proclivity through over exertion, through extent and delicacy of the mucosa, through changes of temperature, through weather, through air pollution, through kind of diet, through change of latitude, through nervous sympathy, through debilitation of the lung tissue, through suppression of perspiration, through a high dew point, through bacteria and other germs. through youth and change of habits.

These are among the most frequent and grave of all affections of the domestic animals. They are especially important however in the case of animals that depend on the soundness of their wind. In horses and dogs accordingly any permanent injury to the organs of respiration will seriously impair the value, not only because of the diminished usefulness of the affected animal, but also because of the probable deterioration of their progeny. The rapid paces demanded of these animals and the strain to which the respiratory organs are subject are potent causes of respiratory disorder. In all animals, however, the extent of the respiratory surface and its extreme delicacy and tenuity especially predispose it to disease. Hales estimates that the mucous membrane covering all the air sacs and air cells is, in the calf, no less than 250 square feet. As the chest of the horse is at least double that of the calf, and as it contains much less connective tissue, and is made up of minute air cells from $\frac{1}{70}$ — $\frac{1}{200}$ inch in diameter and separated from each other by walls so attenuated that the contained capillary blood-vessels are equally exposed to the air on both sides, in two adjacent air cells, the estimate for the average horse must be considerably above 500 square feet. This membrane, incomparably the most delicate and susceptible in the animal economy, is constantly in contact with the air in all its variable conditions, and is necessarily affected by these variations.

The severe changes of temperature are not without their influence on this sensitive membrane. If these changes are sudden, as for example in our northern states where the temperature will vary from 50° to 70° Fah., in a single day, the danger of injury becomes imminent, and the lungs require to be strong indeed to resist their effects. Sudden transition from the hot close atmosphere of the barn or stable to the chilling winds of winter is

equally hurtful. But it is not alone the transition from warmth to cold that is injurious. The general relaxation attendant on the sudden change from a cold bracing atmosphere to one unduly hot is even more injurious. How frequently do human beings suffer from colds as the result of a close sultry period at once supervening on a clear cold one. How extensively do chest diseases prevail among horses brought from the clear pure atmosphere of the field, and shut up in close, hot stables. Here, no doubt, there is superadded the impurity of the too often infected air, the change of diet, of exercise and of general care yet we find that affections of the air passages are to a great extent in ratio with the heat of the building. Hence their constant presence in dealers' stables where it is thought desirable to keep the horses warm to hasten the improvement in the coat.

The suddenness of the transition is usually a principal cause of injury. Where the climate changes slowly the animal economy becomes habituated to it and resists successfully the injurious influences. Thus when spring merges gradually into summer and autumn into winter, diseases of this kind are far less frequent. But on the other hand a sudden and extreme variation of temperature, whether in the ordinary course of the season or from a wide change of latitude, is notoriously attended with diseases of the air passages. Ayrshire, shorthorn and Jersey cattle, when first imported into the Northern States of America, contract colds, consumption and other chest diseases to a far greater extent than the native races, though their progeny or even they themselves after acclimatization, exhibit powers of resistance nearly equal to the native stock. Sheep that have been shorn in midwinter or early spring often repay the inhumanity of their owners by dying of inflamed lungs. Southdown and Leicester sheep, sent from England to the north of Scotland, demand at first the greatest care to protect them against the increased rigor of the climate. The army veterinary statistics of France show that the horses transported from the southern parts of the country to the more northern stations, suffer largely from pulmonary affections. But if the change is effected slowly the requisite powers of resistance are acquired and the novel conditions of life cease to be injurious. That this varied power of resistance is not confined to the higher animals would appear from the

experiments of W. Edwards on cold blooded animals. He subjected them in winter and in summer respectively to a very low temperature and found that whereas in summer their temperature declined 3° to 6° Cent., in winter they had a greater resistance and barely declined $\frac{4}{10}$ ths of a degree.

The action of cold on the surface of the body often leads to morbid states of the air passages as the result of nervous sympathy. A beast is subjected to a keen cold wind, is attacked with shivering, and inflammation of the chest supervenes. The result is rendered more certain if the wind is associated with rain and if the animal has been previously in a state of perspiration. A heavy coat of hair, a profuse perspiration, and a cold draught often combine effectively to produce respiratory disease.

It must be added that the chilling debilitates the nuclei of the animal tissues, and lessens their power of resistance to noxious influences. The excess of cold in the freezing of a part, is followed by congestion and even violent inflammation with perhaps sloughing after it has been thawed. The persistence of such tissue debility is familiar to us all in the example of chilblains. A less extreme application of cold affects the tissues and nuclei less powerfully, but none the less surely. The increased liability to disease of the chilled system is strikingly illustrated in the experiment of Pasteur with anthrax. The chicken which had proved refractory to an ordinary dose of anthrax virus, was dipped in water at ordinary temperature until the heat of its body was reduced, and then it fell an easy victim to the anthrax bacillus. In the same way the person who recklessly exposes himself to wet and chills falls a ready victim to intermittent or yellow fever from which he would otherwise have in the main escaped. Debility from another cause, such as bruise or laceration, favors deep-seated invasion by pus cocci, and a resulting abscess, from which the patient would have remained free, but for such traumatism.

But the effect of cold is not confined to the sympathy between the skin and respiratory mucous membrane, nor the revulsion of the blood toward internal organs, nor to the debilitating of the tissues. The application of cold constricts the vessels and lessens the freedom of the circulation and suppresses the normal cutaneous exhalation. A somewhat similar condition may be in-

duced by prolonged exposure to the rays of a burning sun, the skin becomes hot, dry and rigid, and incompatible with the maintenance of the respiratory function. In either case there is a retention of effete and deleterious matters in the circulation which it was the function of the skin to have eliminated. The danger of such retention may be best exemplified by noting the result of the complete repression of perspiration in the remarkable experiments of Fourcault and Bouley. The former covered dogs and other small animals with an impermeable varnish which induced death after some days or in some cases in a few hours. Bouley shaved three horses and covered the skin with tar. There resulted dullness, torpor, deep, slow breathing, weak and diminishing pulse, muscular tremors, manifest cooling of the body and expired air, and deep violet color of the mucous membranes. They died respectively on the seventh, ninth and tenth days. A fourth horse covered with a layer of strong glue and then with tar perished nine hours after the application. The bodies were like those of animals that had died of suffocation. The mucous membrane of the stomach and bowels was gorged with black blood, the lungs violently congested—dark red and heavy—the air-tubes filled with frothy material, and the lining membrane of the heart had dark spots of blood extravasation. It is no longer then matter for surprise that temporary suppression of the insensible perspiration should be followed by diseases of the chest or abdomen, that extensive burns of the surface of the body should be speedily followed by inflammations of internal organs or that extensive and severe cutaneous inflammations should be associated with internal lesions.

Since the days of Hippocrates it has been universally acknowledged that moist seasons and localities are less salubrious than dry ones. As already observed moisture in a cold atmosphere intensifies its effect. In a hot, close atmosphere it strongly conduces to putrefaction in dead organic matter, and the air becomes loaded as a consequence with noxious gases, and in its lower strata with bacteria in a state of active growth. This condition is most intense in close, unventilated stables, and manifestly operates both in predisposing to and exciting those diseases of the chest and other parts, so frequent in such places. Winds

raise and carry such germs, but also sooner rob them of virulence. (See Zymotic Diseases). Susceptible, young animals, newly housed, usually suffer the most severely from these injurious conditions. Often in their case frequent, extreme and sudden changes, and great atmospheric impurity, are combined with a diet to which they have been hitherto altogether unaccustomed. In young horses there are superadded the exertions—too often extreme—connected with training or work. There are the heats and chills, the soaking perspiration and the frigid winds and rain, the general exhaustion, but particularly the overwork of the respiratory organs, each of itself calculated to superinduce disease. Percivall justly remarks that among young horses, newly stabled and put to work, the prevailing diseases are “catarrh, sore throat, strangles, bronchitis, pneumonia and pleurisy.” His tables of the diseases attacking the horses of his own regiment (1st Life Guards), are so instructive that I here reproduce them :

A TABLE (COMPILED FROM EXTRACTS FROM A “REGISTER OF SICK HORSES” LIMITED TO A GIVEN PERIOD) SHOWING THE COMPARATIVE AGES AT WHICH HORSES APPEAR MOST DISPOSED TO CERTAIN ORGANIC DISEASES.

	No. of Patients under 5 Years.	No. in their 5th Year	No. above 5 Years and under 10.	No. 10 Years and Upwards but under 20.	No. 20 Years and Upwards.	Total.
Disease of lungs ----	170	50	20	50	10	300
Disease of the bowels	10	20	40	70	20	160
Disease of the brain	4	2	5	14	2	27
Disease of the eyes--	30	10	70	35	5	150

It will be seen that nearly one-half of the sicknesses, occurring among the horses of the regiment, were chest diseases, and that nearly three-fourths of these were in animals under five years old, or in those newly purchased from the country.

The subjoined table shows the relative prevalence of disease in different months of the year, deduced from the Register above referred to :

	Disease of the Lungs.	Disease of the Bowels.	Disease of the Brain.	Disease of the Eyes.
January -----	20	12	1	10
February -----	25	8	--	9
March -----	23	11	1	7
April -----	19	10	6	10
May -----	13	3	3	9
June -----	14	16	1	13
July -----	13	13	3	19
August -----	11	23	3	17
September -----	11	5	10	19
October -----	24	3	3	9
November -----	19	10	3	9
December -----	16	9	1	4
Totals -----	208	123	35	135

In this table the extraordinary prevalence of lung diseases in spring and autumn is very noticeable. There only remains to notice the number of deaths occurring in the same regiment from pulmonary and other diseases.

Deaths from pulmonary disease -----77

Deaths from other diseases (Glanders and Farcy
and accidents excepted) -----57

It is thus seen that though individually less dangerous than many affections of the abdomen, brain, etc, yet by reason of their greater frequency, chest diseases induce the greatest mortality among this class of stock.

In treating of the diseases of this class of organs they will be sub-divided according as they affect the *nose*, the *throat*, the *neck*, or the *chest*.

DISEASES OF THE NOSE.

EPISTAXIS. BLEEDING FROM THE NOSE.

Epistaxis as a primary and secondary affection. Causes—mechanical, over-exertion, blood pressure, new formations, diseased mucosa, disease of the nasal venous plexus, disease of heart or lungs, in blood diseases, in hæmorrhagic constitution, in bacteridian diseases of the respiratory organs. Symptoms. Often one nostril, blood bright red, clotted; sneezing, (not retching, acid, nor cough.) *Treatment.* Mechanical, astringent, cold, plugging in solipeds and other animals; hæmostatics.

As a primary affection this occurs more frequently in the horse than in any other domestic animal, though as a symptomatic disease it is common in all farm animals.

Causes. The most common causes are mechanical injury of the Schneiderian membrane, violent congestion of this membrane during extraordinary excitement or exertions, as in coughing, in a closely contested race, in a trying hunt, in drawing heavy loads, especially if uphill and with a tight collar. It may coincide with congestion of the brain, acting to some extent as a vicarious discharge, or with the formation of new structures as polypus, or cancer, in which, from the looseness and friability of their texture, the vessels readily give way. The softened membrane is equally liable to laceration or rupture during the progress of inflammation and particularly when fibrinous (croupous) exudations are being detached. In all these cases animals of a strong, vigorous constitution and with a full or plethoric habit are most liable to be attacked. Various congestions of the mucosa in diseases of the heart or lungs are additional causes. Disease or injury of the cervical branch of the sympathetic nerve, and varicosity of the pituitary venous plexus must be accepted as occasional causes.

Epistaxis is also met with in states of general weakness and with deteriorated blood, as in anæmia, in the course of various fevers and in those hæmorrhagic constitutions in which the altered blood appears to find an easy passage through the debilitated or ruptured coats of the blood-vessels. Thus it is seen in petechial fever in the horse, in *small-pox* in sheep, in anthrax, and in *swine plague* and hog cholera. *Hering* records the case of a num-

ber of pigs suffering from a scorbutic affection and which bled profusely from the nose. In bleeders (hæmophilia) and in leucocythæmia it is liable to appear.

The ulcerations of the mucous membrane occurring in *glanders* and *chronic catarrh* have proved exciting causes of the hemorrhage. Lastly the intense heats of summer and prolonged exposure to the direct rays of the sun induce a general relaxation and a determination of blood to the surface which rouses to activity the latent tendency.

Symptoms. The bleeding, usually from one nostril only, falls in a succession of drops, (rarely in a stream), collects in clots around the nostril, and bespatters surrounding objects as it is expelled forcibly in sneezing. It is usually of a bright crimson hue or, in fevers or poisoned conditions of the blood, of a dark or blackish color. It is distinguished from pulmonary hemorrhage by the absence of cough and of a frothy condition, and from bleeding from the stomach by the absence of the blackened clots and acid odor which indicate the presence of the gastric juice.

It is usually to be further distinguished from these in all animals, save solipeds, by the absence of blood in the mouth.

Treatment. Nasal hemorrhage often stops spontaneously, but if the discharge is profuse or long continued, and especially in weak or anæmic conditions it must be treated energetically. Care should be taken, however, to ascertain first, whether it is not vicarious of some other and more dangerous condition like cerebral congestion.

The head should be placed in an elevated position by tying it up to the rack, and cold water or ice kept applied over the head and neck. Hot water (110° F.) will equally constrict the bleeding vessels. Matico powder may be blown into the affected nostril during inspiration, or a solution of alum (4 drachms to 1 pint of water) or other astringent may be thrown in by means of a syringe. A tablespoonful of peroxide of hydrogen thrown into the nose with an ordinary syringe will give immediate relief. (Gillette.) Antipyrine solution (10:100), Lugol's solution adrenalin (1:1000 locally, 1½ dr. internally), lime salts, or gelatine may be substituted.

Plugging the affected nostril, with a pellet of tow covered with matico, tannin, tincture of chloride of iron (1:10 or 20) or

other astringent may be employed when other means fail. By means of a cord attached to the plug it may be withdrawn after all danger is past. In solipeds, if both nostrils must be plugged, wrap the tow around two elastic caouchouc tubes and introduce these, or in the absence of these perform tracheotomy.

Any tendency to recurrence may be met by the internal administration of gallic acid (horse and cow $\frac{1}{2}$ —1 drachm), acetate of lead (horse and cow $\frac{1}{2}$ —1 drachm), lime salts, or, in anæmic conditions, tincture of the perchloride of iron (horse and cow $\frac{1}{2}$ oz.) in water.

RHINITIS. CORYZA. NASAL CATARRH. COLD IN THE HEAD.

Coryza in the horse. Causes : wet, cold after perspiration, damp climate, stable, soil, new buildings, hygroscopic building materials, youth, age, poverty, nervous sympathy, local irritants, iodine, specific disease poisons. Symptoms, dry congestion, watery discharge, muco-purulent discharge, eyes involved, chill, fever, circulatory and breathing disturbance, defecation, urine, glandular swelling. Inflammation of the sinuses, the severe effects. Duration in slight cases, in severe, in sinus complication. Treatment, hygienic, nursing, dietary, steam, sulphur dioxide, febrifuges, insufflation, electricity, solvent, antiseptic, stimulant.

Under this head will be considered simple inflammation of the nasal mucous membrane. This disease might be considered as a mild febrile affection with the local manifestation in the nose, but it is more convenient to treat of it here as a malady of the nasal chambers.

CORYZA IN THE HORSE.

The chief *causes* are exposure to wet and cold and especially when the subject is exhausted and the skin relaxed and covered with perspiration. In these circumstances a piercing wind, a cold drizzling rain, or a draught in the stable is particularly dangerous. Sudden alterations of temperature and especially a change to a warm stable when the general effect is aggravated by the impurity of the atmosphere and the irritant emanations from dung and urine. Damp climates are more injurious than those that are clear, dry, and bracing, and equally so are damp stables whether the moisture is due to the nature of the soil, such as a cold impervious and undrained clay, or of the building which, from its newness may retain a dangerous amount of moisture in the plaster or because of the hygroscopic properties of the building materials which draw moisture from the surrounding soil. It mainly attacks young horses after they have passed out of the hands of the breeder or dealer, and have been placed in new conditions of life alike as regards feeding, stabling and work. Old and ill-conditioned animals are more susceptible than the strong and vigorous, and the changes of the coat in spring and autumn prove strong predisposing causes. Nervous influence is potent in

causing engorgement of the erectile tissue covering the turbinated bones, and local irritants, like septic dust, lime, ipecacuanha, pollen of certain plants, smoke, and irritating fogs may precipitate it. Iodine in large doses produces temporary catarrh. The weakness of the mucosa from a previous attack predisposes to a second. Bacteria attack the inflamed mucosa. Occasionally the disease sweeps over a country, assuming the form of an *epizootic* when it may perhaps be preferably considered as a catarrhal fever, strangles or mild type of *influenza*, which see.

Symptoms. In the *milder forms* of *coryza* the symptoms may be almost exclusively local, consisting in redness and dryness of the membrane lining the nose, and sneezing, soon followed by the bilateral discharge of a thin transparent watery liquid, succeeded by a turbid flow (epithelial cells in excess) and after two or three days by a thick, white, flocculent, puriform fluid (suppuration, diapedesis). With the supervention of the purulent discharge, comes an abatement of the local inflammation and the freer the discharge the greater usually is the relief obtained and the more rapid the recovery. The eyes are commonly red and watery and sometimes the eyelids are swollen. This implies the continuity of the inflammation through the lachrymo-nasal duct, and the obstruction to the flow of tears into the nose.

When *constitutional disturbance* exists, a rough or staring coat appears as one of the first symptoms, the sneezing is more violent, the nasal mucous membrane is more reddened and swollen, the eyes more dull, sunken and watery, the mouth hot and clammy, the temperature of the body raised, the pulse more frequent and having a sharper beat, the impulse of the heart may often be felt by applying the hand to the chest just behind the left elbow, the appetite is fastidious and the secretions of the bowels and kidneys are diminished, the latter being denser and more highly colored, from the absorption of irritating or infecting matters, the glands under the throat are swollen and the swelling of the mucous membrane may be such as to impair breathing and even to threaten suffocation. In severe cases in which the inflammation extends to the nasal sinuses there is heat and tenderness over the forehead and the pain and weight are manifested by the pendent head and the red sunken, watery eyes and tumefied eyelids. When it extends to the throat, the cough, the difficulty in swallowing and the local tenderness on handling are characteristic.

Course. With the occurrence of suppuration, improvement commences and if the inflammation does not extend beyond the nasal chambers, and if it is not kept up by a repetition or continuance of the cause, the disease will have terminated in recovery in eight or ten days. For ulterior consequences in bad cases see *chronic catarrh, conjunctivitis, abscess of the nasal sinuses, laryngitis, stomatitis, staphylitis.*

Treatment. In slight cases the simplest treatment only is required. Place the animal in a dry, airy, loose box, clear of draughts, and with uniform temperature of 55° to 60° Fah., if obtainable. In the cold season, blanket warmly, and hand-rub and loosely flannel bandage the legs. Feed on sloppy bran mashes only and add half an ounce to an ounce of powdered nitrate of potash daily. Give fresh water *ad libitum*, solicit the action of the bowels by giving injections of warm water three times a day, and encourage the nasal discharge by causing the patient to inhale steam for half an hour or an hour twice daily. This may be done by giving scalded bran in a nose-bag or by keeping the head over a bucket containing hay with boiling water poured over it, the steam being meanwhile directed by a bag open at both ends one of which is fixed around the animal's nose and the other round the mouth of the bucket. As a local astringent, tonic and antiseptic, the fumes of sulphur (burned behind the animal and no more concentrated than can be breathed with comfort) will do much to cut short the attack. It is more soothing if combined with steam. Shut doors and windows, add a few drops of alcohol to some pinches of sulphur and burn on paper laid on a clean shovel or piece of sheet iron. When enough has been used extinguish by covering with a cup or other object. Repeat several times a day. Under this treatment recovery may be completed in three or four days.

In severe cases attended with fever, besides the above a dose of laxative medicine may be given (three or four drachms of aloes), with this precaution, that if the fever is of a low type or the malady epizootic, half the dose only can be safely allowed (2 dr.) on account of the danger of superpurgation. The nostrils must be more assiduously steamed and linseed tea may advantageously replace fresh water as a beverage. If there is much swelling and tenderness of the glands a poultice should be applied to the

throat and between the jaws, and sulphur fumes as advised above, or anodyne astringent insufflation powder may be resorted to. Morphia chlorate two grains, bismuth nitrate, six drachms, and finely powdered gum arabic three drachms may be blown into the nostril during inspiration, or the astringent anodyne injection advised below for chronic catarrh may be used. As introductory to astringent and antiseptic injection, spray, or snuff, the passages may be cleared of their muco-purulent coating by an alkaline solution such as Dobell's:—Sodii bicarbonatii 1 drachm, sodii biboratis 1 drachm, acidi carbolic 1/2 drachm, aquæ 1 pint, mix. This may be injected through a tube syphon into each nostril in turn, or it may be applied in the form of spray. When the surface has been cleansed in this way, the astringent antiseptic dressings reach and act on all parts uniformly and more effectively. Cocaine spray (2:100) with anti-pyrine (5:100) is often very helpful, or the same agent may be used in the liquid form on cotton wool inserted in the nasal chamber. If this is without effect a weak continuous current of electricity will cause constriction and give prompt relief. It may be repeated every few hours. In the absence of this, the emanations from a weak solution of ammonia or from carbonate of ammonia may be used. In cases with excessive and persistent muco-purulent discharge, with presumptive infection from outside sources, or in the young, from the diseased maternal passages, insufflation with calomel, painting with a two grains to the ounce solution of nitrate of silver, or injection with some other germicide may be resorted to.

In case the fever is of a low type, liquor of the acetate of ammonia (4 ozs.), sal-ammoniac (1/4 oz.), or even carbonate of ammonia (1/2 oz.), may be given several times a day, with sweet spirits of nitre (1/2 oz.) and tincture of gentian (1 oz.) Alcoholic stimulants are often used. Inhalations of iodine and iodide of potassium with ether and chloroform are often successful.

SIMPLE CORYZA.

SIMPLE CORYZA IN CATTLE.

Coryza mild in cattle. General treatment. *Coryza* in sheep from exposure, intemperate seasons, clipping. Acute and chronic. Wholesale treatment in flocks. *Coryza* in pigs. *Coryza* in dogs, simple, secondary. Treatment, food, laxative, febrifuge, nauseating, expectorant, antiseptic, gaseous, electric.

This is usually a very simple malady when confined to the nasal chambers, and not of infective origin. When, on the other hand, it attacks the sinuses it becomes a disease of extreme gravity. (See catarrh of the Sinuses). Symptoms are as seen in the horse, but the discharge may be overlooked because of the animal licking it out with his tongue. Treatment does not essentially differ from that laid down above, and recovery may be expected in seven or eight days. If a laxative is wanted give from one to two pounds Epsom salts.

SIMPLE CORYZA IN SHEEP.

Coryza is usually slight and is manifested by sneezing and running from the nose. It occurs in animals clipped or badly sheltered or shipped by rail during the more inclement seasons. In the worst cases the discharge becomes persistent and emaciation ensues so that it is necessary to interfere. Valuable animals may be treated on the same principles as oxen, and in the case of large flocks, by shelter in a warm, dry, clean and airy place and fumigations of steam and the fumes of burning sulphur repeated daily, together with nourishing diet, such as boiled barley or other grain, and quarter ounce doses of nitre and common salt.

CORYZA IN THE PIG.

Hogs are not very subject to this disease and are easily treated by warm, sloppy food, and as a laxative three or four croton beans, according to size, powdered and given in the aliment.

CORYZA IN DOGS.

Dogs are rarely the subjects of simple coryza, though it is constant in distemper. It sometimes proves troublesome in pup-

pies and old dogs. A laxative ($\frac{1}{2}$ —1 ounce castor oil) may be followed in strong and very feverish cases by tartar emetic ($\frac{1}{4}$ — $\frac{1}{2}$ grain) three times a day. Spraying or sponging the nose with a weak solution of chlorate of potass, common salt, or potassium permanganate will greatly relieve. Inhalation from burning sulphur, or from carbonate of ammonia, or both, may be used when sponging or spraying is difficult. In inveterate cases, the weak electric current sent through the cheeks, or the insufflation of acetanilid, iodoform or calomel may be tried. As a rule, saltpeter in five grain doses, given in the water, will prove helpful, and in weak conditions wine, tincture of gentian or nuxvomica may be used.

CROUPOUS RHINITIS IN CATTLE AND HORSES.

A pseudo-membranous inflammation of the nasal mucosa has been especially noticed in *cattle* (Frank, Bollinger, Kitt,) and under the same general conditions as in laryngeal croup. It is characterized by inflammation of a very high type, with extreme redness, many punctiform ecchymoses, a rough, uneven surface destitute of its natural gloss, and, in 2 to 5 days, patches of false membrane, varying in thickness up to about a centimeter. Breathing is greatly accelerated, snuffling, or altogether oral. There is high fever, especially if the morbid process extends to the eye, larynx, lungs or intestine. In some cases it has followed breathing of smoke and hot gases in stable fires; in other cases it has coincided with malignant catarrh or other affection. For general treatment see *Croup*.

As occurring in *horses* it has been attributed to irritant gases inhaled, but as in cattle is probably microbial, at least to this extent that the microbes determine and maintain the character of the morbid process. The inflammation is intense, with deep red, blood-streaked, petechiated, excoriated, swollen mucosa, and a free discharge, yellowish, greenish yellow, grayish-red, or blood-stained, and sooner or later the presence of shreds of false membrane. The case is to be managed like croup, with the advantage that the diseased surface is within easy reach.

CHRONIC NASAL CATARRH. NASAL GLEET. OZÆNA IN THE HORSE.

Chronic catarrh in horse, simple form, loss of tone, inflammation, nature of discharge, glandular swellings, differentiation from glanders. Treatment, astringent tonic, stimulant, hygienic, locally astringents, antiseptics, injections.

A chronic discharge from the nose is often seen in the horse as a sequel of *coryza* or *sore throat*, or as an attendant on other affection of the upper air-passages, and the different conditions productive of this symptom may here be noticed.

1ST. SIMPLE NASAL CATARRH. NASAL GLEET. OZÆNA.

In long standing *coryza* the nasal mucous membrane becomes relaxed, fails to acquire its lost tone and continues to pour out a muco-purulent product. This is really a persistence of inflammation of a low type, under the influence of which the membrane secretes pus in place of its normal mucus. The discharge is white, thick, creamy, has little tenacity, and flows uninterruptedly. There may be slight enlargement of the submaxillary glands, and if the case is of long standing and the patient in low condition, sores may appear on the mucous membrane. These ulcers are distinguished from those of *glanders* by the absence of the unhealthy angry aspect and excavated borders of the latter, by the absence of the small nodular deposits on the mucosa, by the less viscid nature of the secretion, and by the absence of submaxillary swellings, or if these exist, by their being less nodular, less indurated and less firmly attached to surrounding parts. The coincidence of ulcers and submaxillary swellings is always, however, matter for the gravest suspicion, and such cases should, as a rule, be subjected to the mallein test. (See Glanders and Farcy).

Treatment. In simple nasal catarrh, due alone to the relaxation of the mucous membrane, the internal use of tonics and the local application of astringent solutions to the nose rapidly restore the parts to a healthy state.

Among *stimulants*, cubebs, cayenne pepper and copaiba have a stimulating and styptic effect on the mucous membrane and each

of these has been successfully used in such cases. *Cantharides*, in five grain doses, have proved even more successful, (Vines, Percivall). *Sulphate of Copper* in drachm doses in mucilage night and morning has proved very efficient (Sewell, Percivall). *Arsenious Acid* has been employed with still better results. The dose, of five grains, may be intimately mixed with a scruple of bicarbonate of soda and given daily in food.*

But the most efficient tonic in these cases is arseniate of strychnia. Its good effects may be secured by combining with the above mentioned powders of arsenious acid and bicarbonate of soda, half a drachm of powdered nux vomica for each dose.† These powders will usually be taken in food, and may be continued for a month, or until the discharge ceases.

In all cases the general health must be carefully attended to. Keep the patient in a dry, clean, airy building without draughts of cold air; give moderate exercise in the open air; and good grooming; and allow nutritious food of mildly laxative properties,—as occasional bran mash and roots in winter and succulent grasses in summer.

Local Applications. These are the most important remedial measures, and usually of themselves succeed in reëstablishing a healthy condition.

The agents proving most useful are of an astringent nature, and in obstinate cases one may be substituted for another as the last appears to lose its effect. Sulphate of zinc, sulpho-carbolate of zinc, or sulphate of copper in the proportion of half a drachm of either to a quart of water, may be used, or if there is much fœtor, a solution containing a drachm each of carbolic acid and carbonate of potash in a quart of water is to be preferred. In either case the addition of an ounce of pure glycerine renders the lotion at once more soothing and more efficient. The solution must be

* In giving this agent, any redness or watering of the eyes, or colicky pain should be carefully watched for, and when these premonitory symptoms of poisoning are noticed the medicine should be at once suspended to be commenced a few days later in smaller doses.

† Whenever nux vomica or its alkaloids, strychnia or brucia, are given, increased irritability and nervousness should be carefully watched for, and especially any involuntary twitching of the muscles. On their appearance the agent must be suspended and commenced a few days later in half the former doses.

rendered tepid before injecting it, to obviate the irritation attending on the contact of a cold fluid with the delicate membrane of the nose. Among other agents may be named creolin, creosol, creosote, acetate of lead, potassium permanganate, and silver nitrate. Peroxide of hydrogen may be used either as injection or in spray.

The mode of injection is a matter of no small moment. It has been done in some instances by means of a large syringe but the irritation attendant on such a process is an insuperable objection to its use. A better instrument is that introduced by Professor Rey of Lyons. It consists in a tube bent on itself at an angle of 35° so as to form two arms of unequal lengths. The longer fifteen inches in length, one and a half in diameter and widening into a funnel at its free end ;—the shorter about five inches long and tapering towards its free end where its aperture is only two-thirds of an inch across. The instrument is made of block tin or extemporaneously of gutta percha. Over the shorter arm is placed a tightly fitting leather ring four and a half inches in diameter on which is applied some wet tow to adapt it to the nostril and effectually close it. The nose having been drawn in so as to place the head in a vertical position, the short arm of the instrument is introduced into the affected nostril, and the liquid being gently poured into the long arm rises slowly in the nose until it is filled and the liquid flows from the nostril on the opposite side. In introducing the tube, care must be taken that it may not irritate the inner wall of the nose on the one hand, nor pass into the blind pouch, known as the *false nostril*, on the other. A Davidson syringe may be employed.

The greatest gentleness and tact are requisite in thus injecting the nostrils, though in troublesome animals it is sometimes necessary to resort to blindfolding or even to the application of a twitch on the ear, or finally to strapping the animal (head included) to a smooth, firm vertical surface (operating table).

2D. COLLECTION OF PUS IN THE NASAL SINUSES.

Nasal Sinuses, position, orifice, suppuration, symptoms, treatment, tonics, astringents, antiseptics, trephining, significance of the factor, mode of recovery.

In severe *coryza* the nasal sinuses become implicated as shown by the intensity of the symptoms, the prostration, the hanging head, and the heat and sometimes tenderness between the eyes and immediately beneath them on the side of the upper jaw. These sinuses are large spaces filled with air, situated between the superficial and deep plates of the bones of the face and opening into the nostrils by a narrow orifice in the upper part of the nasal chambers. When pus is largely formed in these it fails to flow out as rapidly as produced, parts with a portion of its liquid elements, increases in consistency and sometimes even undergoes decomposition, so that the discharge from the nostril has a putrid odor. Sometimes salts, etc., are precipitated around a nucleus forming a nasal calculus (rhinolith). (Kitt).

The most distinctive symptoms of this form of *nasal gleet* are obtained by percussing the sinuses, and in those cases in particular in which the accumulation is confined to one side of the head, the contrast between the two sides is unmistakable. By gently tapping the forehead with the middle finger from one eye to the other the flat solid sound on the diseased side is easily distinguished from the clear drum-like resonance on the healthy one. By tapping on the bone beneath each eye and just above the maxillary crest the difference between the two sides will be recognized in the same way. In some old-standing cases increased tenderness and slight bulging of the bones over the affected sinuses are often superadded to the other symptoms. In others the pus escapes freely and the sinus is still resonant on percussion. The eye on the affected side is usually retracted so as to seem smaller.

Treatment. In some cases the use of tonics and astringent injections as recommended for the treatment of *ozæna* will prove successful, but more usually it is needful to open and inject the sinuses.

For this the following articles are required : scissors, a knife,

forceps, a trephine or circular saw from half to three-fourths of an inch in diameter and a whalebone or metallic probe.

The horse is thrown and made fast with the diseased side of the head uppermost. A point is then selected on a line drawn between the centres of the two eyeballs and an inch to one side the median line of the forehead ; the hair is closely removed with the scissors, and a semi-circular flap of skin over an inch in diameter is dissected from the bone and turned back toward the poll. The trephine is next applied on the bone and a circular portion, having been cut through, is pulled out by the forceps, when the imprisoned pus will commonly ooze from the opening. A second point is chosen just above the lower end of the maxillary spine already referred to, and opposite the third molar tooth, counting from before ; the hair is removed as before, a flap of the skin raised upward and backward and the bone trephined to open the second sinus. The point of election for this orifice is more important than that of the first. If it is too near the eye, the lower part of the sinus, which is separated from the upper by an imperforate bony plate, is not opened and may continue to keep up the discharge from the nose. If on the contrary it is made too low down, the lower sinus only is opened and the upper being imperfectly washed out from the wound in the forehead will keep up the discharge. Either then this plate must be struck with the trephine or it must be afterward perforated to secure a favorable result. The probe introduced by the wound in the forehead should further appear at the lower orifice.

The cavities are to be washed out first with clear tepid normal salt solution and thereafter daily with an astringent solution such as that used for injecting the nose. If the discharge does not escape freely by the lower orifice its exit may be facilitated by drawing a tape through the sinuses, from the upper to the lower, and retaining it there by a knot on each end.

Marked fœtor of the wound will usually indicate necrosis at the edge of the wound, and demands the use of bone forceps or chisel to remove the offending bone.

A cure is effected by the restoration of the membrane to its natural state, or in other cases by the filling up and obliteration of the cavity by granulation.

ABSCCESS OF THE FALSE NOSTRIL OR TURBINATED BONES.

In young horses as the result of injury from the bridle or severe *coryza*, a circumscribed swelling sometimes appears on the outer flap of the nostril, at first firm, hot and tender, with a surrounding pasty infiltration, then forming into a tense elastic ovoid mass, the size of a pigeon's or chicken's egg. It may become chronic and remain for an indefinite period comparatively insensible to touch and only slightly interfering with the movements of the nostrils. As soon as the elastic tension betrays the presence of pus it should be evacuated by a free crucial incision made from inside the nostrils and the wound plugged with medicated tow and allowed to heal by granulation.

The turbinated bones are two fragile bony structures attached to the outer wall of each nasal chamber. The posterior half of each bone closes the corresponding nasal sinus; the anterior half is rolled upon itself as a sheet of paper might be, and is accordingly open along one side. In this latter a collection of pus may result from severe inflammatory action and the resulting discharge may become somewhat chronic. The flow is greatest after the nose has been raised, from the pus having previously gravitated into a sac in the lower end of the bone. The pus may moreover pass backward into the larynx from the raising of the head and induce a violent fit of coughing. Sometimes the inflammation has extended to the bones covering the nose which are bulging and tender. The thin turbinated bone gives way under the distension, bulges into the nose, and often stops the passage of air through that side. This symptom and the appearance of the swelling cause a close approximation in symptoms to nasal polypus. The facts that it supervened on a severe *coryza*, that it fluctuates on pressure if within reach of the finger, and that pus escapes when it is punctured, exclude the idea of polypus.

Treatment. Puncture of the abscess inside the nose, plugging and daily astringent injections will usually rapidly cure. Gamgee, Jessen and others, recommend trephining of the bone above the nose and washing it out daily, adding that an extensive removal of the bone will correct any existing bulging and deformity.

NASAL DISCHARGE FROM CARIOUS TEETH, ETC.

Ulceration into sinus from caries, loss of molar, overgrown molar. Fœtor, tenderness. Foreign body in the nose.

In cases of a diseased molar tooth in the upper jaw, food getting firmly impacted in the hollow space, irritates the pulp in the fang and the adjacent bone until the progress in ulceration reaches the nasal chamber or sinus and a nasal discharge is established. If an upper molar tooth is lost the molar formerly opposed to it in the lower jaw grows out and sets up the same train of symptoms. In all cases then in which nasal gleet is associated with much fœtor and with difficulty in eating, a careful examination of the teeth should be made. (See Diseases of the Teeth).

FOREIGN BODY IN THE NOSE.

Professor Gamgee records the destruction of an animal for glanders in which the cause of the discharge was afterwards found to be a phlegm ball coughed up into the posterior part of the nose and firmly impacted there.

COLLECTIONS OF PUS IN THE GUTTURAL POUCHES.

Structure, position and opening of pouches, inflamed by extension. Symptoms, discharge intermittent with pendent head, swallowing, pasturing, cough, roaring, dyspnoea, inhalation of food, parotid swelling, glandular enlargement. Nature of contents. Treatment, pasturage, blister, tonics, irrigation, puncture, injection. Concretions in guttural pouch.

Though this is commonly a result of severe sore throat or strangles, yet as it causes a chronic discharge from the nose liable to be confounded with those properly due to diseases of the nasal chamber, it is noticed in this place.

The guttural pouches are two mucous sacs peculiar to solid-peds. They lie side by side above the throat, and in direct contact with the lower surface of the superior bones of the head and the first bone of the neck. They are, properly speaking, dilations of the Eustachian tubes which in all animals establish a communication between the pharynx and the middle ear. The opening into the pharynx is at the anterior extremity of the pouch and close to the posterior opening of the nose, hence the discharge takes place chiefly or exclusively when the head is lowered, since gravitation then favors the escape of the fluid.

Frequently implicated in severe sore throat the walls of the guttural pouches pour out pus as readily as other mucous membranes in a state of inflammation. As the escape of this product is hindered alike by the narrowness of the orifice and, in the elevated position of the head, by gravitation, it frequently becomes imprisoned and inspissated and proves a permanent source of irritation and discharge. In the early stages the contents are glairy with whitish or yellowish clots; later they are creamy, caseous or even cretaceous. The mucosa, at first red, congested and tumefied, becomes in chronic cases, hard, thick, puckered and adherent to adjacent structures. It sometimes ulcerates and the contents escape in mass, through the pharynx and nose, or externally behind the angle of the lower jaw. In the last case water swallowed may escape through the opening. More commonly the pus remains pent up, and thickens, and may dry and roll into round or oval pellets from the movements of deglutition.

The discharge may be arrested for weeks or months when such masses block the outlet.

Symptoms. The nasal discharge is intermittent or irregular, being often partially or wholly suppressed by keeping the head elevated, and reappearing or becoming profuse when it is lowered. Feeding from the ground, nibbling roots, or pasturing, increases the discharge, as the dependent position of the outlet, the jerking and shaking of the head and the movements of deglutition all favor its exit. Swelling of the parotidean region, a flatness instead of resonance on percussion, and the flattening and discharge and sometimes gurgling by manipulation are characteristic. There is cough, roaring during active exertion, sometimes dyspnœa, and, in bad cases, food may be drawn into the bronchia with serious and even fatal results. When the orifice is blocked and the pouch filled with gas the elastic swelling and resilience are characteristic, and pressure may flatten it with a gurgling sound. These symptoms serve to differentiate it from peripharyngeal abscess. The submaxillary lymphatic glands are usually swollen but less than in glanders and not so hard.

Treatment. In mild and recent cases in which the contents of the pouch have not yet become thick and dry, a cure may be effected; *in winter* by feeding the animal from the ground and largely with roots; *and in summer*, by turning out to grass. In either case the matter is allowed to escape almost as soon as formed and the irritated membrane tends to resume its healthy functions. This result will be favored by giving a course of tonics as recommended in *simple ozæna*, and the application of a mild blister to the throat.

Should this fail an operation must be resorted to. Gunther, of Hanover, uses an instrument in the form of a tube a yard long, half an inch in diameter, slightly curved for two inches at one end which is blind, and having an orifice on one side close to this extremity. This tube having been introduced through the chamber of the nose on the affected side and its curved end having been carried into the narrow opening of the Eustachian pouch, tepid water is pumped in and the pouch thoroughly cleaned out. Astringent solutions are then employed. The introduction of the tube is, however, a very difficult operation and one quite impossible to any one who has not the most accurate knowledge of the parts in question.

A second mode of operating is by external incision. For this purpose are wanted scissors, knife, artery forceps, iron probe bent in the form of the letter S, and a tape. The horse having been thrown and fastened and the head extended, the hair is removed from a surface in front of the prominent border of the first bone of the neck, and an incision made between this border and the parotid gland. The incision is made immediately beneath a tendon which may be felt as a flattened cord crossing the border of the bone in its upper third, and it should be carried downward one and a half inches parallel to the margin of the bone. In this preliminary stage the operator has to carefully avoid injury to the parotid gland and the posterior auricular artery and vein. The skin and facia having been divided the index finger of the left hand is pushed inward and forward until the prominent angle of the large cornu of the hyoid bone is felt, together with the muscle (stylo-hyoid) inserted into this bone above the angle referred to. The next step is important, since crossing on the inner side of this muscle and bone at their point of union is the (internal carotid) which becomes subsequently enveloped in a fold of the membranous wall of the guttural pouch. The slightest variation in the position of the artery may here prove fatal unless the greatest caution is used. With the knife guarded by the index finger of the right hand the muscle is cut through from behind forward and the pulsation of the artery felt for beneath. Avoiding its position the knife, with its cutting edge turned forward and its point directed toward the horse's nose, is pushed through the walls of the sac. Or the pouch may be reached and opened by boring with a blunt probe, or the point of a pair of closed scissors. The curved probe is now introduced and carried downward until it is felt beneath the skin just behind the angle of the lower jaw. This may be safely cut down upon with the knife as important parts (vessels and nerves) have been turned aside by its pressure. A tape attached to the probe is now drawn through the pouch and retained by a knot on each end. Tepid water must be injected through the lower orifice daily for three weeks, astringent antiseptic injections thrown in occasionally and the horse fed from the ground. At the end of this period the tape may be removed, and the wounds allowed to heal. During the course of treatment it is always advisable to

change the tape several times by cutting the knot off one end of the old one, stitching the new one to it and drawing it through.

Puncture of the pouch at its lower part is a very simple operation when the accumulation of pus is abundant and chronic. The distended pouch gravitates downward, largely separating the parotid from the deeper vessels and nerves, and finally fluctuates toward the lower end of the gland. In extreme cases it even opens and discharges. When fluctuations can be felt the sac may be incised with a bistuory or abscess knife and treated like a common sore. Opening with a pointed or olive-shaped cautery has the advantage of checking hæmorrhage and securing more perfect drainage. When there is no fluctuation the incision must be made just beneath the lower border of the parotid, the parotido-auricularis being first cut through, then the gland dissected from the deeper parts when the distended sac can usually be felt and opened. If not felt at once it can easily be reached by a careful dissection upward through the loose sub-parotidean connective tissue, with the finger nail, the handle of the scalpel, the blunt probe or the point of closed scissors. A free opening may be made and the wound injected daily with a weak antiseptic solution.

CONCRETIONS IN THE GUTTURAL POUCHES.

Hard cretaceous masses have been met with in the guttural pouches. Dellagana met with an extraordinary case at Bankok, Siam. The subject was an aged Australian mare which rejected both food and drink through the nose. She stood in great distress, fore legs wide apart, head and neck extended, could not lower the head nor turn it to one side. Dyspnœa great. Parotidean region very hard, resistant, swollen and tense. Temperature 101° F. No cough. Was treated with the view of hastening suppuration and pointing, but in a few hours the patient suddenly dropped dead.

"A *post mortem* examination, made the following morning, revealed lesions of intense inflammatory action. The tongue (root and dorsum), post nares, pharynx and larynx were alike affected. The guttural pouches were literally crammed with concretions and pus, which, on removal, filled an ordinary stable bucket of

about three gallons capacity. In size the concretions varied from that of a pea to a hazel nut. The pus was thick and creamy and not putrid. The lungs were congested and the mucous membrane dark in color. The immediate cause of death, no doubt, was asphyxia."

There was no history of the case further than that she had been in active service as a trooper up to within a few days of the visit of Dr. Dellagana. (*Jour. of Comp. Pathology*, Sept., 1898).

DEGENERATIONS AND NEOPLASMS IN THE HORSE'S NOSE.

Hyperplasia. Nasal fibrous polypus, connection, form, size, bony distortion, obstructed breathing, abrasion, ulceration, sloughing, sub-mucous polypus, structure, degeneration. Symptoms: sneezing, snuffling, discharge, palpation, bony swelling, tenderness. Treatment; forceps, hook, ecraseur, knife, saw. Actinomycosis. Myxoma. Sarcoma. Carcinoma. Consistency, structure, fœtor, glandular swelling. Treatment. Recurrence. Fatty tumors. Bony tumors: cancellated or compact tissue, localized or extended. Cysts. Strongyli. Angioma. Bone in pig's nose. Varicosity. Color, obstruction to breathing, hæmorrhage, cicatrization.

These are essentially surgical diseases yet as they are connected with Chronic Catarrh they may be profitably noticed here.

I. Hyperplasia of Nasal Mucosa in Horse. Fröhner, Dieckerhoff and Grawitz describe an abnormal growth and degeneration of the nasal mucosa, affecting especially the lower portion of the nose, which had its mucosa reddened, thickened, roughened, and elevated in nodular swellings the size of a linseed, barley grain or hazel nut, aggregated so as to cause an extensive elevation. These were yellowish red or grayish red, and covered with a grayish catarrhal product. On microscopic section it showed a fibrillar and cellular tissue, highly vascular and showing fistulous vacuoles containing epithelium as of the distended mucous cells, covered with scally epithelium where it was not ulcerated, next was a connective tissue layer, and finally a firm, cell-infiltrated zone which with methyl-violet gave the rose-colored reaction of amyloid. The essential cause of the hy-

perplasia was not discovered, but the tendency was to amyloid degeneration, as found in infected or cachetic states of the system.

II. Fibrous Nasal Polypus. These are connected to the mucosa by a pedicle, or broad base, and vary in size from a pea to a mass which fills the entire nasal chamber, projects from the nostrils and presses outward the septum and facial bones. At times they weigh one or more pounds. They may cause whistling or rattling in breathing, or may completely obstruct the passage of air on the affected side. In time they may cause bulging or even attenuation and perforation of the bony walls, projecting through the hard palate or on the face. Sometimes the surface becomes the seat of granulation, ulceration, or sloughing, causing more or less foetor. The large polypi make their main growth forward and backward, moulding themselves to the form of the chamber, and displacing the turbinated bones. They commence to grow under the mucous membrane and as they grow and become more loosely attached they carry this as an outer covering and pedicle. When incised they show a structure of interlacing bundles of fibres, with cell elements more or less abundant, according to the rapidity of growth. Gravitz found amyloid degeneration of the walls of the blood-vessels and mucous follicles, and of the fibres.

Symptoms are difficult breathing, snuffing, a smaller current of air on the affected side, or none, sneezing, a watery, purulent, bloody, or foetid discharge, and the appearance of the polypus when the nasal chamber is examined in a good light. If beyond reach of vision the polypus may often be felt by the finger. Care must be taken not to mistake the red, angry surface of the turbinated bones in Catarrh for a polypus. If beyond the reach of the finger, the flat sound on percussion of the nasal and frontal bones on the affected side, and the persistently diminished flow of air may serve for diagnosis. Tenderness shown on percussion is common to this and abscess of the sinuses.

Treatment. The horse having been cast with the diseased side uppermost and the head turned to the light, the tumor is seized with the fingers, the forceps, or hook, and drawn gently outward. The chain of the *ecraseur* may be passed over it and slowly tightened upon the pedicle until it is cut through. This will usually obviate any laceration of the turbinated bones and consequent

bleeding. In case of serious hæmorrhage check by cold water, ice, the actual cautery, or by plugging and styptics. Polypi with a broad base may be removed with a probe-pointed knife, curved on the flat, and furnished with a long handle. The mass is seized with a vulsella and detachment made by passing the knife with the concave side toward the tumor. In cases where the tumor cannot be seen or reached, some have resorted to slitting up the outer wall of the nostril as far as the angle of union of the nasal and maxillary bones, care being taken to make the incision outside the upper end of the cartilage of the ala nasi. If too high to be satisfactorily reached in this way the nasal or frontal bone may be trephined over the body of the tumor as indicated by the flatness on percussion, and the operation performed through the opening thus made.

III. **Actinomycosis.** Though much more common in cattle than horses, yet the occasional occurrence of this in the face of the soliped must not pass unnoticed.

IV. **Myxoma of Septum Nasi.** This is a mucous degeneration of the cartilage with destruction and liquefaction of the tissue and the formation of mucous cysts, embracing the whole thickness of the septum and standing out in each nasal chamber, of the size of a grain of barley to a hazel-nut. These have been frequently found in the *horse*, and have been described by Leisering, Müller, Bruckmüller, Siedamgrotzky and Kitt. *Myxoma* has also been met with in the submucosa of the nose in *cattle*, and *fibro-myxoma* in the horse.

Myxo-malacia (Kitt) of the turbinated and facial bones leading to thickening and closure of the walls of the outlet into the nose is a cause of **dropsy of the antrum** with distension downward to obstruct the nasal passage and upward causing bulging, attenuation and absorption of the maxillary, molar, and lachrymal bones. Relief of the tension can be had by perforation of the wall of the sinus through the turbinated or maxillary bone, but the morbid process in the bone remains.

V. **Sarcoma, Osteosarcoma and Carcinoma.** These are found growing from the periosteum, or even starting in the cancellated tissue and projecting into the nose, where they give rise to symptoms like those of fibrous polypi. Being much softer in texture and more liable to ulceration and degeneration they are

likely to cause a much more offensive discharge. There is also more tendency to the implication of the submaxillary lymphatic glands. The only treatment is surgical and recurrence is always to be feared. (See Diseases of the Orbit.)

We have found them filling the nasal sinuses, and again extending through the maxillary plates into the roof of the mouth.

VI. **Fatty Tumors** of the nose are described by Röhl, Gurlt, Bruckmüller, etc., as existing on the septum and in the sinuses. Being simple, they can be removed with great confidence as to non-recurrence.

VII. **Osseous Tumors of the Nasal walls.** These are described by Röhl as osteophytes in the maxillary sinus in chronic catarrh, and by Gamgee as osteomata attached to the outer wall of the nasal chamber, which had to be detached by saw and bone forceps. I have found these latter of a soft porous structure easily detached by the knife, and in other cases dense, and requiring chisel, saw and forceps. In one instance the tumor grew from a dense hypertrophy of the maxillary bone which could not be entirely removed because the molar alveoli were implicated.

VIII. **Cysts** named by Röhl and others as present in the mucosa of the ethmoid cells in solipedes often contain larva of the *sclerostoma equinum*.

IX. **Angioma** may be but an exaggerated development of the abundant venous plexus and erectile tissue on the surface of the turbinated bones. There appears to be at other times an actual increase of the vascular tissue. As might be expected it has no abrupt margin, but gradually shades off into the healthy tissue. The prominent centre has a bluish red or brownish hue. It obstructs breathing, is apt to bleed under violent exertions in draught, or in contested races, and readily ulcerates with a bloody discharge. If it subsides and heals, it is followed by a whitish puckering like the so-called *cicatrix* of glanders.

FOREIGN BODIES IN NOSE OF PIG.

In the *Annales de Médecine Veterinaire* for March, 1904, Lenard records the case of a pig with snuffling breathing, which disappeared when both nostrils were closed compelling the animal to breath through the mouth. There was no sign of osteo-

malacia of the facial bones nor any appreciable thickening or softening. The pig finally improved but was very paretic in its limbs.

When killed it presented in the nasal chambers 5 or 6 particles of decomposing, loose, bone, averaging over an inch long by over half an inch thick. They had led to destruction of the mucosa, submucosa and septum nasi so that right and left nasal chambers communicated. There were found gray hepatization of the lung, ulcers and false membranes of the ileum and exostosis on the articular extremities of the bones of the limbs.

The bones had evidently been forced into the posterior nares in coughing or vomiting, and had they been recognized might possibly have been washed out by a stream of water passed through the nose from behind or in front.

In the horse a bolus given medicinally is at times coughed up into the posterior nares, giving rise to a chronic nasal catarrh.

CATARRH OF THE FRONTAL SINUSES IN CATTLE. CATARRH FROM TRAUMATISM.

Extent of sinuses in cattle. Causes, blow, unequal teams, locking horns, fracture. Pathology. Congestion, exudation, suppuration, swelling and closure of outlet, prostration, fever, agalactia, septic infection, ulceration, exclusion of oxygen. Symptoms. Crimson hæmorrhage, disturbed breathing, appetite, rumination, position of head and eyelids, percussion and temperature of forehead, fever. Chronic form. Slow progress, emaciation, anorexia, facial expression, hide, discharge, breath heavy or foetid. Duration. Prognosis. Lesions in sinuses and glands. Treatment. Cold irrigation, icebags, elevation of head, laxatives, diuretics, dehorning, trephining, injections, astringent, antiseptic, blister, tonics.

The gravity of this affection is a consequence of the great extent of the delicate mucous membrane which lines the frontal sinus. This cavity not only occupies the whole forehead from beneath the eyes up to the frontal crest, but extends in the mature horned animal, into the tapering bony process which forms the basis of support for the horn. The mucosa is rendered all the more extensive by the numerous pillars and septa that pass from the outer bony plate to the inner, giving great strength to the part for purposes of offense and defence. Inflammation of this membrane is usually the result of blows on the horns, and these are much more common among working oxen than dairy cows. The immediate cause is violent contact with the yoke when the head is lowered at pasture, and from blows of a club in the hands of the driver. In countries where the yoke is a broad padded board hung from the horns and resting on the forehead traumatic injuries are much more common. The active and vigorous animal gets the greater part of the work, and the wrench and jar may induce hæmorrhage and catarrh. If the yoke is ill-made or badly fitted the case is worse. The blows sustained by horn or forehead in an ordinary fight, may also be the cause, and a partial or complete fracture of the bony support is especially hurtful when the detached horn is replaced so as to close in the cavity. Blows on the frontal crest are also dangerous.

The *pathology* of the disease consists in an inflammation of the mucosa of the sinus, and the filling of that cavity with blood or, later, with a muco-purulent fluid, the escape of which is pre-

vented by the closure of the nasal outlet by swelling. This of itself produces violent headache and much nervous disorder as witnessed by the drooping head, closed eyelids, prostration, high fever, anorexia, and in cows suppression of the milk secretion. But there is reason to believe that this is aggravated by the septic germs, which inspired with the air, were already present in the sinus, and which in the comparative absence of oxygen, in a rich culture medium and in contact with injured and debilitated tissues, assume an enhanced pathogenic rôle. This may serve to explain the ulceration of the mucosa of the sinus found in subjects that have suffered for some time. It further explains the notorious fact that the free access of air (oxygen) to the inflamed sinus is one of the most helpful therapeutic measures.

Symptoms of the Acute Form. When the disease is traumatic the first symptom is usually a hæmorrhage from the nose, the blood being of a bright crimson. Respiration is hurried, and appetite diminished, yet rumination may be imperfectly performed. The bleeding may be repeated for days in succession, but the ox is still capable of work. On the fifth or sixth day there is complete anorexia, rumination ceases, the head sinks, resting on the manger or soil, the ears droop forward and downward, and may be swollen. The head inclines to the affected side, the corresponding horn is intensely hot, and the eyes are closed. Light percussion of the forehead on the affected side gives pain, and the sound elicited is flat and dull as compared with that from the opposite side. The temperature of the body rises 2° or more, the pulse becomes frequent, full and hard and the impulse of the heart abnormally strong. Costiveness, partially suppressed and high colored urine, and dry hot muzzle betray the fever. Unless relieved the chronic form may supervene.

SYMPTOMS OF THE CHRONIC FORM. When this comes on slowly, working oxen get emaciated, lose appetite, have the eyes dull and sunken, and the lids drooping, the coat rough and staring and the skin harsh, dry, and lacking in pliancy, the head is carried low when out of the yoke and, after shaking the head and sneezing, a glairy, slightly foetid matter escapes from the nostril. The breath is foetid and appears to be offensive to adjacent cattle.

This may continue for months with no other change than a more constant nasal discharge, and increasing emaciation and weakness.

Prognosis. This is favorable for the acute disease at the outset. But if no relief is furnished it is liable to go on to a fatal issue. Even the chronic form is curable unless the subject has already become hopelessly weak and debilitated.

In *fatal cases* the sinuses are found to be filled with a glairy fluid and the mucosa thickened and raw or ulcerated. There may be enlargement of the pharyngeal lymphatic glands, and there may be attendant pharyngitis.

Treatment. The patient must have absolute rest and cold water irrigation or icebags applied to the head. The bowels may be opened by a saline, or a diuretic administered. If the head is persistently dropped it may be kept moderately elevated by a halter tied to a higher point. Should there be no relief at the end of twenty-four hours, no time should be lost in securing free admission of air to the cavity. Cruzel advises to saw off the horn at its base, as the one certain method of securing prompt improvement and speedy recovery. If a horn and its bony support have been broken off they should be at once removed and the head turned up to evacuate the accumulated glairy fluid from the sinus. From an apparently hopeless condition, a few hours will suffice to restore an appearance of good health. If the horn has not been broken and it is desirable to save it, the bone may be trephined in front of the root of the horn and the liquid evacuated, or less effectively and more painfully the horn may be bored at its root by a large gimlet.

If no hæmorrhage has taken place and if active treatment has been adopted at the outset, recovery may be complete in two or three days, but if the disease has been ushered in by a hæmorrhage which recurs several days in succession, amputation of the horn or trephining will be demanded. In chronic cases this should be followed by astringent and antiseptic injections and a blister may be applied to the throat or the side of the neck. In these cases too a course of mineral tonics is desirable.

CHRONIC CATARRH OF CATTLE.

Catarrh, Chronic, summer aggravation, thickened, roughened mucosa, discharge, twigs in nose. Question of parasitism. Treatment, remove causes, antiseptic astringents.

A remarkable form of chronic catarrh with summer aggravation exists in some of the hilly districts of New York but has not received such study as to enable us to state its true nature.

One or two in a large herd will have a loud snuffing breathing, which may subside so as to be entirely overlooked in winter, but reappears when put to pasture in the spring and continues in a marked form throughout the warm weather and until after the animal is returned to winter quarters. There appears to be little or no fever nor constitutional disturbance except what comes from the obstructed breathing, and the yield of milk may be unchanged. The symptoms would indicate a purely local disease. Yet so few are attacked out of a herd that it cannot be actively contagious.

On close examination the nasal chambers are found to be narrowed, there is a manifest thickening of the mucosa, and its surface feels rough and uneven, with miliary elevations. There is of course more or less glairy discharge. If the examination is made about midsummer, the finger introduced into the nose will usually detect the ends of twigs that have been introduced into the cavity and broken off. When withdrawn these may prove individually from four to eight inches long, and some force may be required to extract them. In winter these are often absent, having been apparently dropped one by one. The absence of these sources of irritation sufficiently accounts for the manifest improvement during the colder months. In spite however of the winter remissions the disease tends to a steady advance year by year. While nothing definite is known of its pathology, the occurrence of this disease in given localities, its manifestly local nature, and its persistence when once established would suggest enquiry as to the possible existence of parasitism, bacteridian or otherwise.

Until further discovery, treatment can only be of a general nature. Removal of the foreign bodies from the nose, pasturage

where there is no brush to replace them, soiling when clean pastures cannot be found, and the use of astringent and antiseptic agents by insufflation or injection would be indicated.

Great improvement amounting to temporary recovery has been secured by a daily smearing of the whole nasal mucosa with $\frac{1}{2}$ ounce compound tincture of iodine in a pint of castor oil. This is introduced on a pledget of cotton on a wire or whalebone staff. It should be continued for a week or two.

MALIGNANT CATARRH OF CATTLE.

Local causes. Debilitation. Polluted air. Poor diet. Symptoms, gastro-intestinal, fever, lachrymation, turbid aqueous, photophobia, congestion of mucosæ, generally disturbed circulation and breathing, depression, heat of forehead, buccal petechiæ, epithelial desquamation, abrasions, ulcers, abortion, albuminuria, local swellings, shedding of horns, dropsy, dyspnœa. Lesions, in nasal mucosa, subcutem, cerebral, dark blood. Prognosis. Treatment, antiphlogistic, laxative, diuretic, tonic, locally steam, antiseptic, astringent, trephining.

This disease occurs chiefly in cold damp marshy localities where the vital power is impaired or in cold situations exposed to severe north and east winds. In the wet cold seasons of spring and autumn it is especially prevalent. According to *Rychner* it rarely attacks old cows but prevails among young cows and oxen. In the south of France on the contrary it appears chiefly in the hot season (June and July) and is attributed to suppressed transpiration. It prevails especially however in herds kept in small filthy stables, low in the roof, hot, close and badly aired. (Festal). In New York it appears in cattle on black muck pastures and in Minnesota on the dried up ponds.

Symptoms. Diarrhœa is a common premonitory symptom arising from the irritation of the intestinal canal as it is soon followed by some degree of costiveness, the dung becoming dark colored, firm and scanty. Diarrhœa reappears later. The coat stares or the beast actually shivers; the head is depressed; the roots of the horns and the forehead are hot; the eyes are sunken, swollen and red, suffused with tears, turbid in their anterior chamber (aqueous humor) and intolerant of light; the muzzle dry and hot; the mouth hot but moist with abundant saliva; the mucous membranes of the mouth, nose and vagina have a bluish red color; the pulse is rapid and more or less full or hard; the impulse of the heart weak; the breathing is accelerated, the respiratory sound is heightened in intensity and a cough is frequent. Temperature 104° to 107°F. The urine is scanty and high colored. The surface of the body is alternately hot and cold, and after some time a watery fluid begins to distil from the nose.

At the end of twenty-four hours the symptoms are intensified or altered. The eyelids are more swollen and the flow of tears more profuse; the nasal discharge becomes slimy, and streaked with blood, and accumulations take place in the frontal sinuses as indicated by the increasing heat of the forehead and the dullness on percussion. In the mouth appear dark red spots, from blood extravasation, over which the epithelium sloughs off leaving raw unhealthy sores. The appetite entirely fails; dung and urine are passed painfully and with effort, and abortion frequently takes place in pregnant cows. The urine is albuminous with cell forms, and casts. The limbs appear rigid and it pains the animal to move.

From the fourth to the sixth day the ulceration appears on the mucous membrane of the nose which has often a claret color, and the nasal discharge becomes again more watery and irritating. The muzzle is swollen and a dropsical infiltration appears beneath the jaws, which extends along the neck to beneath the thorax and into the limbs. Portions of the nasal mucous membrane now slough off, and similar sloughs are often seen on the skin of different parts of the body; the secreting structures of the horns and hoofs even participating, so that these are easily detached or shed. Saliva flows profusely from the lips, a fetid watery diarrhoea succeeds the constipation, the dropsy becomes nearly general and death occurs on the eighth, ninth or tenth day of the illness. Convulsions and symptoms of suffocation may precede death.

In a *post mortem* section the principal lesions are found in the nasal cavities and skin. The areolar tissue in both is the seat of an abundant serous infiltration, which has taken place into the deeper layers of the skin as well, rendering it thick, hard and unyielding. Besides the sloughs and ulcerations on the skin and mucous membranes, false membranes have been met with, on the lining membrane of the mouth and air passages. The ulcers in the nose have in many cases reached the bone, and from the abundant infiltration and softening, the membrane is easily stripped from the walls of this cavity and of the sinuses. The general infiltration appears to have reached the brain, which is described as softened and having an undue amount of liquid in its cavities. The blood contained in the vessels is dark colored

and numerous patches of extravasation are visible on the mucous and serous membranes as well as in the interior of organs.

Unless the malady can be controlled in its early stages it usually proves fatal. Patients that recover after it has been well developed sometimes retain its effects in permanent blindness or palsy of the hind limbs.

Treatment. Early and vigorous antiphlogistic measures are strongly recommended by French and Italian veterinarians. Gelle and Ercolani advocate the most copious bleedings. Festal insists that all other measures are useless when this is neglected. Before adopting free sanguineous depletion the history of his practice was a record of deaths, whereas later his losses were in cases where from a failure to recognize the disease at the outset, from the existence of diarrhœa, from the patient being pregnant or from a fear that the milking value might be impaired, bleeding was deferred. He pushed the bleeding to the extent of causing acceleration of pulse, quickened breathing and heaving of the flanks, to effect which sixteen pounds had to be abstracted on an average. If this were done early the engorgement of the muzzle had usually greatly diminished if not entirely disappeared in the course of seven or eight hours thereafter. The alleged benefit is probably largely due to elimination.

Less heroic treatment is now generally adopted. An active purgative (one and a half pounds Epsom salts) may be given even though apparently contraindicated by the premonitory diarrhœa, and a further useful derivation may be obtained by applying active friction or even stimulating embrocations to the legs.

Steam with or without sulphur dioxide may be inhaled as for ordinary *coryza* and cold water or ice kept applied to the forehead.

Nitre in ounce doses daily or liquor of the acetate of ammonia in three ounce doses may be given after the purging has ceased. Or drachm doses of hydrochloric acid with bitters may be given thrice a day in at least a pint of water.

Where the nasal discharge persists after the subsidence of the other symptoms the sinus should be trephined in front of the horn, and tepid water and mild astringent and antiseptic lotions injected until a healthy action has been established. Change to a dry, well drained pasture or building is desirable for both treatment and prevention.

MALIGNANT CATARRH OF SHEEP.

This resembles the corresponding disease of cattle, but cannot as yet be considered identical with it. Its occurrence in epizootic form strongly bespeaks an infection or the taking of toxic matters produced by microörganisms outside the body. There is intense congestion with redness, often ramifying, of the nasal mucosa, conjunctiva, and other connecting mucosæ. The general color of the affected mucosa may be bright scarlet, dark red or dark blue with numerous petechial points implying the action of a deadly hæmolytic poison on the blood. The mucosa is covered with a slimy, creamy or caseous product, and is strongly infiltrated, tense and it may be glistening. Coincident inflammation of other mucosæ, especially that of the bronchia, and also of the parenchyma of the lungs and of the skin, bespeaks a general diffusion of the poison throughout the system as in the cattle affection. *Prevention* and *Treatment* may be attempted along the same general lines as in the ox.

PARASITIC AND CONTAGIOUS DISEASES OF THE NOSE.

COCCIDIAN NASAL CATARRH IN RABBITS. Vol. V.

LÆECHES. Vol. V.

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MOLLUSCUM CONTAGIOSUM. Vol. IV.

PETECHIAL FEVER. Vol. IV.

PINK-EYE. Vol. IV.

S. AFRICAN HORSE FEVER. Vol. IV.

STRANGLES. Vol. IV.

VARIOLA. Vol. IV.

VESICULAR EXANTHEMA. Vol. IV.

AFFECTIONS OF THE THROAT.

Sore throat, Angina, Cyanche—is a generic name applied to a series of inflammatory affections of the various structures about the throat. If the *larynx* is specially inflamed the disease is known as *laryngitis*, if the *pharynx*, as *pharyngitis*, if there are exudations forming false membranes it is *croupous* or *diphtheritic*, or if associated with some general febrile affection, it takes its name accordingly, *influenza, strangles, distemper, or scarlatina*, as the case may be.

CATARRHAL LARYNGITIS IN THE HORSE.

ANGINA LARYNGITIS, CYNANCHE LARYNGEA, ETC.

Definition. Causes, microbial, mechanical, cold, irritants, extension, diet, debility, plethora, domestication, close stables, infectious disease. Symptoms of acute form, head extended, throat swollen, tender larynx, cough, in early stage, after exudation, wheezing in inspiration, dysphagia, fever, œdema glottidis, spasms, dyspnoea, successive discharges. Lesions, tumefaction, softening, friability, redness (ramified or not), erosions of form, chronic form, in old debilitated animals, in those reined too tightly, in those which perspire with difficulty or bear heavy coats. Symptoms, local, in breathing, cough, effect of cold air, or water or of dust. Sequelæ, ossified cartilages, roaring, emphysema, bronchiectasis. Treatment, hygienic, soothing, sheepskin, compress, poultice, mustard, sulphur dioxide, laxative, neutral salts, expectorant, sedative, derivative, tracheotomy, with trochar and canula, with scalpel, tracheotomy tubes. Insufflation, injection. In chronic laryngitis, electuaries, mustard, derivatives, astringents, caustics, tonic inhalations.

Definition. Laryngitis is inflammation of the larynx and most commonly of its muscosa alone.

Causes. These include all the ordinary causes of chest diseases. In all alike microbes fill an important rôle, and these not simply forms that are essentially pathogenic, which produce spreading infections and epizootics, but simple saprophytic germs which are found on the laryngeal mucosa in health, and which only become dangerously pathogenic when the integrity of the mucosa is in some way impaired so that it falls an easy victim to the microbial growth. The lowering of the vitality

and resistance may be attributed to many different causes, some of which operate through the general system, while others have a more local action. Among the former may be named general debility from insufficient or improper feeding or overwork, from impure air, close confinement or darkness, or from stabling in damp underground basements. These act more efficiently in early youth, and in the old and debilitated. Debilitating diseases may pave the way for an attack—indigestions, lung diseases or valvular diseases of the heart, causing the circulation of carbonized blood, nephritis, entailing the undue retention of waste products, or even parasitisms may thus operate. Closely allied to these are chills, cold rains or snows, prolonged exposure without exercise or clothing, or after clipping, or after perspiration and fatigue; or swimming or wading in cold waters. High feeding, hot, close stables, in contrast with the sudden chill when taken out doors, and extension from rhinitis, pharyngitis, staphylitis, guttural pouch inflammation, bronchitis, pneumonia, and even from the infectious strangles, influenza, contagious pneumonia, and glanders must be recognized.

As direct and special causes may be named; Severe pinching of the larynx to cause coughing; spasmodic inhalation of food materials that lodge in the larynx; introduction of a probang into the larynx, in unskillful attempts to pass it by the mouth; and the inhalation of irritating and infecting dust. The larynx is, however, much less exposed to infection than the nasal passages, as irritants and microbes are liable to be arrested by sticking to the moist Schneiderian membrane and thus fail to reach the larynx. In forcible administration of volatile, irritant liquids (ammonia solutions, oil of turpentine, carbolic acid, chloroform, ether, etc.), the larynx is very liable to suffer. In the same way operate irritant gases (dry air of stoves, empyreumatic gases, chlorine, bromine, sulphur fumes, etc.). Krebs mentions a case of laryngitis from contact with infecting pus discharged through the Eustachian tube from a ruptured abscess in the middle ear. Finally the bites of parasites (leeches, *œstrus* larva, *linguātula*) may start inflammation in the irritated mucosa. Fractures of the laryngeal cartilages by violence, punctures, and even surgical operations implicating the larynx may be incriminated.

Symptoms of acute form. All acquainted with horses can recognize the general symptoms of sore throat. The nose is elevated and protruded to avoid compression of the larynx; it is carried stiffly for the same reason. There is some swelling around the throat or beneath the root of the ears. If the cartilages of the larynx are compressed between the finger and thumb, or if pressure is made in the median line below upon the connecting crico-thyroid membrane the patient instantly coughs and throws up the head to avoid a repetition of the suffering. This tenderness of the larynx to touch is peculiar to laryngitis and serves to distinguish it from pharyngitis. The cough is at first very hard and painful and only gives way to a soft mucous type when a free exudation puts an end to the tense, thickened and dry state of the mucous membrane. The inspiratory act is accompanied by a whistling or deep bass sound, particularly after the slightest exertion. This may be heard at times during expiration as well, though not invariably so. Sometimes the animal drops the food from his mouth after mastication, because of the pain attendant on swallowing, but this is really a symptom of coexistent pharyngitis, and its absence implies the nonexistence of that complication. There is usually a slight pasty swelling between the branches of the lower jaw.

There are besides the general symptoms of fever more or less marked, such as increased temperature, accelerated pulse, red injected eyes and nose, slightly hastened breathing, the expiration being effected by a double lifting of the flank as in broken wind (heaves) etc. In one or two days a slightly frothy, serous discharge appears and rapidly encreases.

In two or three days in favorable cases this exudation becomes opaque, white, flocculent, muco-purulent, the cough becomes softer and less frequent, the local tenderness decreases and the general symptoms subside. The hand applied over the larynx detects a fremitus, and sibilant or strident râles are developed.

If exudation delays, the symptoms may become more intense, and breathing may get loud and difficult in connection with thickening and rigidity of the mucous membrane, or a serous exudation into and beneath it (œdema glottidis) which by closing the glottis renders breathing almost impossible. The same distressing symptoms may arise from spasm of the larynx excited by the

inflammatory action. As arising from thickening or infiltration of the membrane these symptoms may come on comparatively slowly, but in case of spasm they appear suddenly and have periods of intermission, reappearing on succeeding days and usually in the early part of the night. In such circumstances the loud, noisy breathing is heard at a considerable distance, the horse stands obstinately still, the fore feet apart, his elbows turned out to allow a freer action of the chest, the flanks working laboriously, the head low, the nose protruded, the nostrils widely dilated, the mouth open, the eyes standing out from their sockets red and wild looking, and the face constrained and pinched, the whole expression being that of intense agony from impending suffocation.

Lesions. In cases where death has supervened, perhaps in connection with another disease, the laryngeal mucosa, especially on and above the glottis, is soft, tumid, friable, with ramified or uniform redness and petechial spots. The epithelial layer may be softened, disintegrated and shed, leaving minute or larger erosions, which are, however, usually superficial. Pressure expresses muco-purulent matter from the mucous follicles. In case of œdema glottidis the mucosa and submucosa are thickened by an abundant exudate which may extend to the connective tissue outside the larynx as well. In aggravated cases there may be dark red or brownish red discolorations of the mucosa. Swelling may involve the vagi nerves explaining stridor.

Course, Duration. Fortunately these aggravated forms of the disease are rare and unless the patient perishes during such an attack or the inflammation extends down toward the chest, laryngitis rarely proves fatal. Its duration is from twelve to fifteen days. Its extension to the lungs may be suspected when the extreme tenderness of the throat subsides without any corresponding improvement to the health. Examination of the chest will then rarely fail to detect the presence of disease.

But although sore throat is rarely fatal its effects are not unimportant nor trivial. It occasionally merges into a chronic form, with a hacking cough, tenderness to pressure and an increased liability to other diseases of the air passages. More frequently it is followed by wasting and fatty degeneration of the laryngeal muscles and the horse becomes a confirmed *roarer*. In all cases it leaves a greater susceptibility to a second attack.

Sub-acute Laryngitis. This form has been chiefly seen in young animals and up to eight years old. At the outset its symptoms are moderate but as it is usually associated with serous effusions in and around the mucous membrane the symptoms above mentioned as indicating imminent danger of suffocation may suddenly appear and life can only be preserved by opening the trachea.

Chronic Laryngitis. This may follow the acute form or it may come on independently and by slow degrees. It may accompany nasal catarrh, or chronic bronchitis. Old animals which have had heavy draught work and repeated attacks of sore throat, are frequent subjects of it, and as Fergusson has pointed out it is most prevalent among horses whose throats have been compressed by the inconsiderate use of the check rein. Reynal has observed it often in horses that are sweated with difficulty, and in those which remain long wet from the length and thickness of their winter coats. It may affect the mucosa, sub-mucosa, muscles or even nerves.

Symptoms. When acute laryngitis passes into the chronic form all the symptoms subside except a slight nasal discharge, the cough, tenderness of the larynx, and *roaring*. The cough is dry, short, and hacking, rarely soft, and is heard mainly when the animal feeds, when he leaves the hot stable for the cold air, and after drinking cold water. During exercise it is equally excited, the cough becoming harder, and the horse extending his head and neck as if to disengage some body from his throat. The subject may in nearly all other respects maintain the appearance of vigorous health.

Course, etc. This disease is liable to prove obstinate and if of old standing, often incurable. Unless checked, the continued congestion and irritation of the larynx, the frequent, hacking cough, and the consequent distention of the lungs bring about extensive and irreparable structural changes. Among these may be mentioned ossification of the cartilages of the larynx; paralysis of the left laryngeal nerve with wasting of the muscles to which it is distributed, and *roaring*; dilatation of the bronchial tubes, and permanent distention and rupture of the air cells (emphysema, broken wind, heaves).

Treatment. Acute form. Unless in the very mildest cases un-

accompanied by fever, repose is essential. If available, a roomy, clean, dry, and airy loose box should be allowed, care being taken to avoid draughts of cold air and to secure a soft equable temperature neither too hot nor too cold. Blankets should be used and even flannel bandages applied loosely to the legs if the weather is cold, or if there is any tendency to chills and shivering. The nostrils must be steamed as directed for *coryza*. A piece of sheepskin with the wool turned in may be tied around the throat and up to the ears. In very acute cases a linseed meal poultice or wet pack may be applied to the throat, while in the milder forms, a mustard poultice or a lotion of Spanish flies or other stimulant may be employed. Unless the malady has an epizootic type, with prostration and a weak, rapid pulse, the bowels may be opened by a laxative (3 or 4 drachms aloes), and the water or gruel the animal daily drinks should contain $\frac{1}{2}$ to 1 ounce saltpeter. As an expectorant the patient may take salammoniac 1 oz. daily in the drinking water, or this agent may be evaporated from a clean chafing dish every two hours and inhaled. Or he may take bicarbonate of potash or soda, or iodide of potassium. If the cough is troublesome, half a drachm of Dover's powder may be given thrice a day or 1 grain chloride of apomorphine every hour. Bromide of potassium or sodium may also be resorted to. Inhalations or spray of sulphurous acid, or vapor of oil of turpentine and insufflations of calomel (in absence of potassium iodide) may benefit as local applications. The diet must be confined to sloppy bran mashes, cut roots, or boiled barley, or oats. Hay should be withheld in the more acute cases until improvement appears. Under treatment such as the above and even without the medicinal part of it, the great majority of cases will do well.

In cases attended with high fever with strong full pulse and bright red nasal membrane, the purgatives and diuretics are especially called for, and the former should have their action encouraged by frequent hot water injections. Twenty drop doses of the tincture of aconite repeated four times a day, or ten drops every three hours, will be useful. Acetanilid may replace this. Ozonized terebène (tallianine) intravenously (10-20 cc. daily) increases leucocytosis (polyneuclear) and proves of great value especially in infective cases.

When the symptoms are of such a type as portend the access of

paroxysms of threatened suffocation, bleeding has been strongly recommended, but unless resorted to in the first twenty-four or forty-eight hours is rarely admissible. Also in weakened constitutions and when the fever is of a low type, with small, weak pulse and general dullness and prostration, the temporary relief obtainable from blood-letting will not often counterbalance the danger of increasing weakness, and the loss of recuperative power. In such cases the application of a strong mustard poultice for several hours in succession, until an abundant effusion has taken place into the skin and beneath it, has often the best effect by virtue of its depletive and derivative action. Active friction of the limbs to improve their circulation and increase their temperature is also useful.

Dieckerhoff injects daily into the larynx a solution of 1 gram iodine, 5 grams iodide of potassium and 100 grams distilled water.

Jelkmann injects morning and night 2 grams chloride of morphine, in 300 grams of bitter almond water, and claims the subsidence of the fever in one day, and complete recovery in a few days.

Anech applies the agents in fine sprays from an atomiser with excellent effect.

Tracheotomy. When suffocation becomes imminent not a moment must be lost in performing *tracheotomy*. This operation is always available in threatened suffocation from obstruction to the passage of air in the nostrils and throat.

Different methods of opening the windpipe have been resorted to. One is by means of a cannula and trochar at least three-fourths of an inch in diameter and about five inches long and with two large oval orifices in the middle of the cannula, and on opposite sides. This is made to transfix the windpipe with its investing skin and muscles from side to side in the middle of the neck, care being taken to pass it in the interval between two adjacent cartilaginous rings. The trochar is now withdrawn and the orifice in the cannula corresponding to the interior of the windpipe, the animal is enabled to breath freely through the tube. The cannula has only then to be secured in its place by a tape carried round the neck.

The more common plan is by introducing a tube through a circular opening made in the trachea. For this operation are

needed scissors, knife with a thin narrow blade, needle and thread, and tracheotomy tube. The common tube is about an inch in diameter, four to five inches long bent upon itself so as to fit into the trachea, and furnished with a flat shield to slits in which cords may be attached to fix it in its place. A second variety is only long enough to reach into the windpipe. It is provided with a flattened shield externally and from its inner extremity there projects downwards at right angles a plate curved so as to adapt it to the form of the interior of the trachea. There is an additional plate to fit into the upper part of the tube, provided with two lips projecting from it at right angles; the outer lip is screwed to the shield after the tube has been introduced into the wind pipe and the inner lip is thus fixed inside the ring of the trachea, immediately above the opening and effectually prevents any displacement of the tube.

In operating, the animal is kept standing with the head as nearly as possible in the natural position. The hair is removed from the skin beneath the windpipe between the middle and upper thirds of the neck. The skin having been rendered tense (without displacement) by the fingers and thumb of the left hand, an incision is made in the median line from above downwards, for about two inches and is carried through the muscles so as to expose two rings of the trachea. The needle and thread are passed through the membrane connecting the two rings, and with the knife a semi-circular piece of cartilage is cut from each of the two adjacent rings. The thread in the connecting membrane prevents them from being drawn in by the rush of air. It only remains to introduce the tube and fix it in position.

Not only does tracheotomy obviate immediate danger of suffocation, but by removing the source of irritation in the continuous and forcible rush of air through the narrowed and inflamed tube, and in securing for the blood a freer aeration and a purer constitution it often induces a rapid change for the better in the character of the inflammatory action. The wound may be daily cleansed and dressed with sodium hyposulphite.

Some veterinarians following the example of Bretonneau and Trousseau have treated sore throat from the first by what is called the abortive treatment. For this purpose a long whale-bone probe with a pledget of tow firmly attached to its end and covered with powdered alum is introduced through the mouth

into the pharynx and larynx even. Violent paroxysms of coughing are induced, but cures are affected in from two to five days. Under Delafond's treatment calves and foals recovered in twenty-four hours. A more modern method is to inject a solution by means of a hypodermic syringe inserted between the upper rings of the trachea.

Milder treatment such as the inhalation for an hour several times a day, of the fumes of burning sulphur and water vapor will be found generally successful. The air should be impregnated with sulphur fumes only so far as can be breathed without inducing coughing on the part of the patient. Such measures should not divert attention from the necessity for general care, a control of diet, clothing, air, the state of the bowels, nor from local external applications to the throat.

Treatment of Chronic Laryngitis. The patient should have a loose airy box with an equable temperature. The avoidance of work and exposure must be sought for the time. Green food, cut roots, boiled grain, or bran mashes, with little or no hay, or other dry food, must be given. The bowels must be regulated. An electuary composed of linseed meal, molasses, and a drachm of belladonna extract to every tablespoonful of the mixture, may be given to the extent of a tablespoonful smeared on the inner side of the cheek twice daily. A mustard poultice to the throat has often a good effect. Light firing over the larynx is sometimes beneficial.

If secretion is defective and cough hard and dry, chloride of ammonium, carbonates or bicarbonates of soda, potash or ammonium or borax, in solution or in gaseous form, may be given, the various bitters being at the same time drawn upon as tonics. If secretion is excessive, with a loose gurgling cough, astringents are indicated like ferric sulphate or chloride, ($\frac{1}{2}$ dr.); or they may be applied as spray; alum or iron alum five grains to the ounce, zinc sulphate or sulphocarbolate two grains to the ounce, silver nitrate one-half grain to the ounce or iodine 4 grains, iodide of potassium 10 grains to the ounce of water. These may be introduced through the nose with the head elevated, or in small genera through the fauces. Tar, oil of turpentine, creosote, carbolic acid or eucalyptol may be inhaled from hot water.

Cadeac succeeded perfectly in two obstinate cases by a seton around the throat.

PHARYNGO—LARYNGITIS IN CATTLE.

Susceptibility. Causes, symptoms, cough, salivation, wheezing, lachrymation, muzzle dry, tender throat, dysphagia, disturbed innervation and circulation, hyperthermia. Duration. Abscess. Treatment, laxative, local treatment, lancing.

Cattle are less subject to sore throat than horses. The skin appears less sensitive to the influence of cold and heat. The ox is not subjected to the same severe exertions. It is rarely seen to sweat, the moisture passing off from the surface as insensible perspiration only. The disease, however, recognizes the same causes as in the horse, though these are manifestly less injurious. Cadeac draws attention to its greater frequency in working oxen ; in cattle fed on swill of breweries and distilleries ; and as occurring from mercurial poisoning ; also to the frequency of a contagious form following specific hæmaturia, attended by high temperature 104° to 107° F., and running a course of two or three weeks. Recoveries appear to be the rule.

Symptoms. The disease usually affects at once the larynx and pharynx so that the symptoms are somewhat modified. In the simplest form there is only a small, hacking cough, a flow of saliva from the mouth and some loss of appetite but no fever. In more acute cases the breathing is loud and wheezing, the cough, soft and rattling, is followed by a free discharge of mucous from the mouth, the nostrils and eyes are red, the muzzle dry, the pulse accelerated and full, the throat tender to the touch, and swallowing difficult, part of the food and drink being rejected through the nose. If the larynx is chiefly involved the loud noise in breathing is the predominant symptom and sometimes almost the only one.

Course, etc. The cough and other symptoms are usually moderated with the access of the abundant secretion on the second or third day, and recovery is perfect on the eighth to the fifteenth. If abscess results, to which there is a far greater liability than in the horse, it may not burst till the twentieth day and the case is correspondingly protracted. This should be carefully distinguished from the deposits of tubercle which take place around the throat in cattle. In rare cases the disease becomes chronic.

Treatment does not differ from that advised for the horse except in the greater safety of purgatives which must in this case be saline (Epsom or glauber salts one to two pounds), and in the greater ease with which local treatment can be applied owing to the shortness of the soft palate. When abscess forms it must be encouraged by poulticing and opened with the knife or lancet as soon as it points.

LARYNGITIS IN SHEEP.

Infrequency. Causes, damp lands, storms, close buildings, clipping. Symptoms, cough, sneezing, discharge, snuffling, oral breathing, tender throat. Treatment, ventilation, warm water vapor, sulphur dioxide, salines.

Sore throat is fortunately even more rare than in the larger ruminants. It occurs chiefly when this animal, constituted to feed on the dainty grasses of the dry mountain side, is kept on cold, marshy ground and exposed to frequent cold, wet blasts. Sheep suffer also from exposure in cars, from hot, close, filthy buildings in winter and from unseasonable clipping.

The *symptoms* are frequent coughing and sneezing, running from the nose, working of the jaws, and breathing through the open mouth as being easier than through the plugged nostrils. The larynx is tender and may be swollen.

Treatment is usually confined to ventilation and cleaning of the fold, frequent fumigation with water vapor from the spout of a boiling kettle, and with sulphur fumes, and giving tepid farinaceous gruels or mashes containing sulphate of soda in the daily proportions of two pounds to each hundred head of sheep. Salammoniac may be given in food or drinking water.

LARYNGITIS IN PIG.

Frequency. Causes, wet, cold pens, exposure, withholding liquids. Symptoms, prostration, dullness, cough, fever, swollen throat and neck, dyspnoea, dark mucosa, sloughing of epithelium and epidermis, general petechiæ, foetid breath, great prostration. Lesions, gangrenous patches on pharynx and fauces, ulcers, infiltrations. Treatment, hygienic, dietetic, emetic, laxative, poultice, bandages, locally, astringent, antiseptic, caustic, tonic.

Sore throat is common in some localities when pigs live in herds.

Causes. Chiefly faulty hygiene. Exposed, cold and wet piggeries, cold blasts for which the pig has an extraordinary aversion, and the deprivation of liquids in warm, dry seasons are frequent causes.

Symptoms. These have been described by M. Pradal, who divides the disease into three stages, evidently dealing with an infectious malady. The *first stage* is marked by loss of appetite, dullness, slow, listless movements, a tendency to hide under the litter; low, hoarse grunt and cough, the last aggravated by moving the animal; pain in swallowing; red, sunken eye, and constipation. If there is no improvement on the second or third day it merges into the *second stage*. This is characterized by a still hoarser grunt, painful, hard, hacking cough, difficult breathing, especially in the sunshine, and a rapidly increasing swelling of the throat, soon extending to both ears and as far down as the breast bone. This engorgement feels soft and pasty though firm, tender lumps may be felt, indicating the approaching formation of abscess. It is so abundant that suffocation may ensue in the course of forty-eight hours. If the progress of the swelling is not arrested it soon passes into the *third* or *gangrenous stage*. The breathing is more hurried; the mouth open, the protruded tongue of a bluish black color, the cough followed by a continuous rattle, the head unsteady, swallowing impossible, and the swelling extends to the side of the face and beneath the chest. The swollen surface is cold and livid; the bristles easily detached; it is bedewed by a serosity which exudes from it, and portions of the dead skin tend to detach themselves. The mouth and throat participate in the gangrene, the breath, saliva and nasal discharge

are fetid, and the epidermis peels off. The snout, ears and skin generally assume a bluish black hue, the prostration is extreme, the creature lying constantly on its side; the pain ceases and in one or two days death ensues preceded by a state of comparative calmness.

On opening the throat after death the mucous membrane is engorged and thickened, bears various hues of black, blue, livid and green, and breaks down into a pulpy mass under a slight pressure. The surrounding (pharyngeal) muscles even are implicated in this change. In the earlier stages there is only engorgement with blood of the tonsils and the mucous membrane of the pharynx and larynx, serous infiltration of the surrounding part, and often the presence of inspissated mucous resembling false membranes or of ulcers on the surface.

Treatment. In the earlier stages, hygienic measures alone may suffice to check. A warm, dry, comfortable piggery, emollient and astringent drinks, such as sheep's head broth, oatmeal and other gruels acidulated with vinegar or buttermilk, an emetic (six grains of tartar emetic); a dose of physic (four croton beans powdered and given in the food, or from two or three ounces of castor oil), and if the patient will permit it a flannel bandage or piece of sheepskin around the throat. If the symptoms are more threatening it is recommended to bleed from the ears and tail; to apply a linseed meal poultice round the throat to hasten the formation of abscesses, or in the absence of such indications employ a mustard poultice made with spirits of turpentine, or rugs wrung out of boiling water, to the same part. Local astringent and caustic applications to the throat are the most promising, applied by means of a whalebone probe as recommended for other animals, the mouth being held open by a noose round the upper jaw. Sodium sulphite, silver nitrate, potassium permanganate, hydrochloric acid diluted, and tincture of iodine, may be employed. Give tallianine intravenously.

When the gangrenous stage has been reached all treatment is useless.

LARYNGITIS IN THE DOG.

Sore throat is chiefly seen in pampered pets and in hunting dogs, as the greyhound. In the latter class it is the result of chills, a cold ducking when heated, cold damp kennels, etc. It is sometimes almost the only manifestation of distemper.

Symptoms. Dullness, impaired appetite, a slight cough roused by exercise, drinking, pressure on the larynx, etc., becoming more frequent and paroxysmal. These paroxysms give rise to accelerated and panting breathing, rubbing the chops with the paw and if severe, to the ejection of a glairy yellow (bilious) material from the stomach. There is also running from the nose and frequent sneezing. The dull muffled bark has lead to the supposition of *rabies* but it has no resemblance whatever to the characteristic cry of *rabies* which beginning like an impulsive bark merges into an agonized and baffled howl. Accelerated pulse, elevated temperature, reddened fauces and swollen tonsils are marked symptoms. Expectoration is usually swallowed.

Sometimes a short dry cough obstinately remains after the disease has apparently subsided. The acute disease may last 8 or ten days.

Treatment. Attend to general comfort, steam the nostrils, give a laxative if costive, and follow with iodide of potassium or salammoniac (5 grains) repeated thrice daily. Apply a mustard poultice to the throat. Astringent, sedative, antiseptic sprays are even more applicable and successful than in the horse. Friedberger and Fröhner advises morphine chloride 1 to 2 decigrams, bitter almond water, and distilled water, of each 20 grams : dose 10 to 15 drops thrice daily. Use tallianine in veins.

PHARYNGITIS. SORE THROAT.

Causes. Symptoms, larynx insensible, tender parotid and sub-maxillary swelling, cough mucous, difficult swallowing with gurgling, liquids returned by nose, buccal mucosa hot and red, salivation, chronic cases. *Treatment,* medicated drinks and electuaries.

ANGINA PHARYNGEA. CYNANCHE PHARYNGEA.

Inflammation of the pharynx owes its existence to the same causes as *Laryngitis*.

Symptoms. The general symptoms being like those of *Laryngitis*, the specific and distinguishing ones only will be here noticed. The larynx is not tender to the touch nor is cough thereby excited. The glands beneath the root of the ears (parotid) are swollen and tender and cough is induced by handling them. The intermaxillary glands are enlarged. The cough is loose and followed by the ejection of glairy materials by the mouth and nose. Food and drink are swallowed with difficulty and effort, dry grain or hay is often refused, or dropped from the mouth, after it has been chewed, to avoid the pain of swallowing. Deglutition is accompanied by a gurgling sound caused by the abundant secretion in the pharynx. In swallowing liquids a portion is often rejected by the nose. The mouth is hot, red at its back part, and filled with fetid saliva which often drips from between the lips in the coarser breeds of horses. The fever varies according to the intensity of the inflammation. This disease is rarely serious, and improvement is manifested, by a free discharge from the nose of a white opaque color, by the ability to swallow without pain, and the better appetite and general appearance. Collections of pus in the *Guttural pouches* may result from pharyngitis. See *Chronic Nasal Catarrh*.

I have seen chronic cases of this disease due to fracture of the large branch of the hyoid bone; and Reynal, to laceration or ulceration of the soft palate, and an abscess of the pharyngeal mucous membrane.

The *treatment* does not differ materially from that of laryngitis except in the greater value of soft food, mucilaginous and acidulated drink and of electuaries which act on the throat as they dissolve. Subjoined is a formula:

Recipe: Extract Belladonna, four drams; potassium iodide, one ounce; sodium hyposulphite, three ounces; mellis, five ounces. Mix. A dessert spoonful to be smeared on the inside of the cheek thrice daily.

CROUP. CROUPOUS (PSEUDO-MEMBRANOUS) LARYNGITIS.

Croup : croak. Acute laryngitis, with spasms and perhaps pseudo-membranes. Relation to diphtheria. Causes, low, damp localities, exposure, youth, form of inflammation. Symptoms, onset sudden, crowing inspiration, hard cough paroxysmal, fever, larynx sensitive, dyspnoea in paroxysms or intermittent. Complications. Duration. Lesions, intense congestion, false membranes on larynx, fibrinous. Treatment, fomentations, ether, chloroform, chloral hydrate, laxative saline, sedative, derivative, surgical, water vapor, calomel, caustic, stimulants, tonics. Croup in sheep and horse.

Name and Definition. The word *croup* by which this disease is known over the whole of Europe and a great part of America is, essentially *Scotch*, and is familiarly used in the Lowlands of Scotland to signify—to *croak*. The disease consists in an acute inflammation or high vascular irritation of the larynx, associated with spasms of its muscles and commonly though not invariably with a firm layer of exudation on the surface of the mucous membrane. In some cases undoubtedly croup is but a form of the contagious pseudo-membranous affection diphtheria, the germs of which grown on a surface freely swept by continuous currents of pure air, retain too much of an ærobie habit to penetrate deeply into the tissues. (See Author's "Malignancy mitigated by Oxygen," Medical Record, 1881, p. 673). It does not follow, however, that croup is always due to even a weakened germ. So far as yet appears it may develop independently of any particular pathogenic germ, from some violent local irritant in a predisposed subject. Croup, therefore, may be treated here as a presumably non-infectious disease. Being a very rare disease in horses its manifestation in ruminants will first be noticed.

CROUP IN THE OX.

Causes. These are not well understood. Low, damp situations would seem most liable, especially if the animals are much exposed at night. So far indeed as can be observed it arises from the same causes as *laryngitis*. Age affects its development. Croup is mostly seen in animals between six months and a year old, and rarely in those over five or six years of age. The specific

cause of the formation of false membranes and of spasms of the laryngeal muscles is a mystery, but to these the susceptible constitution and tissues of young animals appear to predispose. No mere grade of inflammation from the slightest hyperæmia to the highest type of inflammatory action is of itself sufficient to arouse the special phenomena. All of these are seen everywhere but *croup* may be said to be confined to certain localities and ages.

Symptoms. Unless it supervenes on a pre-existent attack of catarrh, croup is usually as sudden in its outset in the lower animals as in man. An extremely hard *croupy* cough, or loud, crowing, difficult breathing, loudest in inspiration, is usually the first symptom and appears to seize the animal in an instant and without the slightest premonition. This is closely followed by intense fever, full, hard pulse, 80 to 100 and upward per minute, increase of bodily temperature sometimes to 107.5° F., costiveness and high colored, scanty urine. The throat is excessively tender, the slightest touch giving rise to violent paroxysms of coughing, during which the eyes redden and protrude from their sockets, the veins of the skin are gorged, the tongue, dry and livid, is protruded and small portions of the contents of the stomach and white shreds of false membrane are occasionally brought up. Sometimes in the intervals of coughing as well the mouth is constantly open and the tongue protruded and partly covered by a frothy but tenacious mucus. Suffocation appears imminent in many cases and the beast may perish suddenly in this way. On the other hand, the threatening symptoms may be present only at certain periods of the day and may be moderated remarkably at others, especially at early morning. If complicated by any chest affection the symptoms are more urgent and the issue more commonly fatal. If associated with a low type of fever, a small, weak pulse, and much prostration, as it tends to be if it continues several days without relief, it has a more fatal tendency. The same may be said of its occurrence epizootically.

Duration. Croup will often run its course and prove fatal in twenty-four to forty-eight hours. Improvement is manifested by the cough becoming less convulsive and painful, by the expulsion through the mouth of shreds of false membrane, and by return of spirits and appetite.

Postmortem Appearance. If the animal has died suffocated, the

lungs and right side of the heart will be gorged with blood; if in a stupor (coma), attendant on brain poisoning with venous blood, the veins will be specially engorged. The mucous membrane of the larynx has a more vivid aborescent redness than in ordinary laryngitis but the special feature is the presence of false membranes. These layers of exuded material are almost confined to the air passages. They may extend to the soft palate and nose in an upward direction and to the trachea and bronchial tubes in a downward, but they rarely exist in the mouth, pharynx, or gullet like the false membranes of diphtheria.

Characters of the false membranes. These are gray or yellowish white, though they may be reddened in patches or streaks. They vary in consistency from that of glairy mucus to a firm layer as of dense fibrine, and become more adherent as they are of older standing. Sometimes they are partially detached, the free end of the shreds floating in the larynx. The deep or attached surface presents redness in points, in streaks, or as ramifications very visible if the membrane is held up between the eye and the light. They vary in thickness from half to a line. Delafond has found these membranes in the lower animals to be mostly formed of fibrine, with a little albumen, and traces of alkaline and earthy salts.

Treatment. This must be prompt and energetic. Wet cloths as hot as the hands can bear, wrapped around the throat and neck, and replaced as they cool, will usually arrest the spasm. If this fails, ether or chloroform by inhalation or chloral hydrate by injection may be employed with caution. The action of the bowels must be secured by salines (sulphate of soda $\frac{1}{2}$ to 1 lb) or oil (linseed oil $\frac{1}{2}$ to 1 pint) and injections of warm water. Sulphate of soda should be thereafter given in half ounce doses twice daily, or nitrate or acetate of potass may be substituted. They are advantageously given in linseed decoction and may be combined with laudanum, ($\frac{1}{2}$ ounce) belladonna, or other agent to check the spasms. Spraying with a solution of cocaine 3:100, or its application on a swab on the end of a whalebone staff, bent at the end, will often relieve the violent symptoms and may be repeated when necessary.

A blister (mustard poultice) should be applied at first either to the throat or breast, the windpipe being left untouched lest

tracheotomy should be required. Similar applications to the legs are useful.

If suffocation appears imminent, *tracheotomy* should be at once performed (see under Laryngitis). This operation has been depreciated because of the late period at which it has been employed, when the patient was already past all hope, but the resulting wound in the neck is more than counterbalanced by the greater freedom of breathing and the better aeration of the blood which tends to obviate the justly dreaded low fever. It often leads to a rapid diminution of the spasms and laryngeal irritation.

Agents applied directly to the inflamed mucous membrane are often requisite. The air of the building should be rather warm, equable and moistened by water vapor, if that can be conveniently done. Calomel or alum powder may be frequently introduced into the larynx by means of a whalebone probe and sponge as spoken of under laryngitis, or a solution of nitrate of silver (10 grains to the ounce of water) may be applied several times a day. These not only hasten the removal of false membranes but counteract their production. They produce violent and convulsive coughing at first and have to be used carefully. Delafond blew in such agents through an opening made in the windpipe. They may be injected with a hypodermic syringe. In prostrate conditions it may be necessary to resort to stimulants (wine whey, carbonate of ammonia) and tonics (gentian, Peruvian bark). Tallianine intravenously may be tried.

PSEUDO-MEMBRANOUS LARYNGITIS: CROUP IN SHEEP.

According to Roche Lubin, croup is sometimes observed in spring in lambs and yearlings. The common cause is "the shutting up of the animals for the whole twenty-four hours in a hot confined place, the floor of which is covered by a fine dust, and the air loaded with the same, owing to the jostling of the sheep together, the effects being intensified by the weight of the fleeces."

The disease is manifested by constant working of the jaws, extreme tension of the neck, abundant salivation, respiration hurried and whistling, extreme pain and threatened suffocation when the slightest pressure is made on the throat, and refusal of all food liquid or solid. The weak, hacking convulsive cough is

associated with the discharge of a whitish glairy mucus by the nose until the third or fourth day when false membranes may be expected.

Treatment is like that for the ox, medicine being given in about one-fifth of the doses.

CROUPOUS LARYNGITIS IN PIGS.

Kept in filthy pens, in impure air, and allowed to lie on, and burrow in masses of decomposing manure, the pig is especially subject to severe attacks of sore throat, and the attendant inflammation is often associated with the formation of false membranes. The sloppy diet given, and the large demand of the healthy porcine system for abundance of pure air, tend to beget in these circumstances a strong predisposition, and the confined unwholesome pens preserve stores of infecting microbes ready to seize upon the debilitated system or tissue, which has thus been deprived of a large part of its resisting power. Once started on a pathogenic career, these microorganisms acquire more and more power of injurious survival in the body, and thus the affection spreads as an epizootic to all denizens of the same pen or establishment. Cold rains or sleet, chilling draughts of air, excessive summer heat, severe thunder storms (electric tension), and all debilitating conditions contribute to the predisposition or become the occasion of an attack. As in nearly all affections attended by microbial invasion, young pigs suffer more than the mature, and high-bred delicate races (Berkshire, Essex, Suffolk, Yorkshire, Chinese, Poland-China, etc.,) show a special susceptibility.

Symptoms. These are dullness, stiffness, inappetence, a disposition to mope apart, or burrow under the litter, a hard, dry painful cough, swelling of the throat, movements of the jaws, and it may be frothing at the angles of mouth. Examination of the mouth shows in the early stages more or less redness, becoming intense as it approaches or involves the soft palate and tonsils. These parts are covered by a grayish or yellowish-white, grumous exudate and a similar material, more or less foul smelling, escapes from the nose and mouth. Very soon, patches of false membrane, grayish or white, are observed on tonsils and palate and detached shreds of this croupous deposit may mingle with the discharge.

The high temperature and hurried, wheezing and even raucous breathing which were marked at the outset become exaggerated, the mouth is held open, the tongue blue, the nose elevated, the prostration extreme, the animal being unable or nearly so to support himself on his hind limbs though making efforts to rise.

This advancing paresis is liable to be mistaken for that attending on swine plague, and the error is all the more likely when scarlet or dark red blotches appear on the skin. These however come later than is usually the case in swine plague, the disease is confined to one herd, kept under specially faulty conditions, and there is no record of an opportunity for the introduction of infection. Finally the concentration of the inflammation on the fauces and larynx, and the presence of the visible false membranes serve to complete the diagnosis.

Course. The disease advances and usually in about four days terminates in recovery or death.

In favorable cases the false membranes are loosened, and expelled during a violent fit of coughing, the breathing and temperature subsides to nearer normal, the eyes become fuller and brighter, the control of the limbs better, the visible mucosæ change from a blue to a pinker hue, and gradually appetite is restored.

In cases that prove early fatal, the pig remains prostrate, with mouth open, tongue protruding and blue, eyes sunken, and breathing dyspnoic, loud and stertorous. Death usually occurs in a paroxysm of coughing. In some cases the affected mucosa becomes gangrenous prior to death, but the patient rarely lives long enough to allow of its separation by sloughing, and of a subsequent healing.

Prognosis. This depends largely on the promptitude and energy of antiseptic treatment. At first the disease is largely local and superficial and the invading microbes are easily reached and destroyed, whereas, later, the microörganisms have encroached on the deeper layers of the mucosa, on the submucosa and subjacent structures, and are practically beyond the reach of antiseptic treatment. Cases early treated may have a very low mortality, and neglected ones a very high one.

Lesions. The infected mucosæ and that of adjacent parts are

infiltrated, swollen, friable, and even in patches gangrenous. The false membranes covering the laryngeal mucosa, are thick non-vascular pellicles, often of considerable thickness, they may be torn or detached so as to be shreddy, and are covered with a layer of tenacious mucus. From the first the mucous follicles are distended and filled with an inspissated mucus. The extensive infiltrations and congestions of surrounding parts may cause great swelling, redness, and doughy sensation in the region of the throat.

Treatment. At the outset an active antiseptic dressing is usually promptly effective. Silver nitrate, 10 grains to an ounce of water, or hydrochloric acid $2\frac{1}{2}$ drams to the ounce, have been mainly resorted to. These should be applied to the fauces, tonsils, pharynx and above all to the interior of the larynx, by a small sponge firmly fixed on the end of a whalebone staff curved at about an inch from the point. A single early dressing is usually effectual, but a second or even a third may be applied if the affection continues. The use of non-poisonous antiseptics (sulphites, hypo-sulphites, salicylic acid, sodium salicylate, copperas, boric acid) in the food, is much less successful because they, in the main fail to reach the affected parts in a sufficiently concentrated condition. Tallianine may be used.

Prevention. Other swine (especially the young) kept under similar conditions should be kept carefully apart from an affected herd, and those not yet affected should be separated, as the microorganisms that have lived in the animal system have become more potently pathogenic. For the same reason the pens and yard should be cleaned and disinfected and even left empty for some time.

No less important is it to change the regimen of the affected herd, placing it under more wholesome conditions of living especially as regards cleanliness, pure air, and protection against exposure.

CROUPOUS LARYNGITIS IN SOLIPEDS.

This is a rare affection in the horse, which has less disposition to the production of false membranes than the other domestic animals. As in other genera, the predisposition is greatest in the young, and hence cases have been mostly seen in foals and young horses.

Causes. The occasion of the attack has in many cases been the partial loss of vitality and resistance of the mucosa of the larynx owing to contact with violent irritants. Riss records two cases, and Bouley one, determined by inhaling the hot gases and smoke in a burning stable. Irritant gases like ammonia have long been blamed for such cases, as has the inhalation of pure chlorine gas. Bretonneau, experimenting with caustic substances injected into the trachea and bronchia (silver nitrate, mercuric chloride solution, cantharidine), succeeded in producing false membranes. It seems, however, that an extreme measure of disorganization of the mucosa is necessary, to lay it open to the microbial attacks which result in croupous products. Cadeac claims cases from the forced administration of very hot drenches.

To a less extent than in other animals, yet no less really, debilitating conditions predispose, such as sudden, extreme changes of temperature, exposure in the open field in cold, wet nights when unaccustomed to this treatment, and even the drinking of extremely cold water.

As in other animals, the microorganisms, formerly enjoying a saprophytic life in the air passages, attack the debilitated mucous membrane and become active pathogenic factors.

Symptoms. Dullness, leaving of food, hurried breathing, dilated nostrils, small, dry, painful cough, soon becoming resonant, strong and croupy, highly injected mucosæ, and very elevated temperature are marked symptoms. The larynx is very tender to touch, and the hand feels a distinct thrill or fremitus, from the vibration of the false membranes. The breathing becomes stertorous (*roaring*), tremors prevade the whole body at intervals, the face is drawn and anxious and the mouth may remain open with the hanging tongue blue or violet. Auscultations of the larynx will now detect a very coarse mucous râle or gurgling which is very characteristic. The nasal mucosa is of a deep red, interspersed with ecchymotic spots, and the nasal discharge is filamentous, yellow and in time contains shreds of the false membrane which may be gray, greenish or blood-stained. The dyspnœa is liable to be extreme at intervals, especially after a cold night, and seems largely spasmodic.

Course. The symptoms may be grave and very threatening for from six to twenty-four hours, yet in favorable cases recovery may take place in three or four days, and as a

rule by the end of a week. In such cases the false membranes have been early detached and expectorated. In fatal cases these membranes remain adherent and increase, the swelling lessens the passage which is still further narrowed, or completely closed, by the attendant spasms, and the animal dies asphyxiated.

Lesions. In fatal cases the laryngeal mucosa is found to be covered with the yellowish layers of false membrane, often of great thickness. Where recent, and only in a thin layer over the deep red congested mucosa, it may have the appearance of a thick glairy mucus, but the older layers are dense, thick, and firm, notched at the edges, and blood-stained on their deep aspect, by which they were attached to the violently inflamed mucosa. The mucosa under the membrane seems rough, abraded, and even at points ulcerated, the surface presenting many pus corpuscles. The membrane is fibrillar with many leucocytes and nuclei, and, in its deeper layer, blood globules.

Treatment. The affection being somewhat less deadly than in other animals, it may often subside without the same actively antiseptic treatment, yet that treatment when it can be applied is eminently satisfactory. Milder measures, usually adopted consist in sodic sulphate in the drinking water to the extent of gently relaxing the bowels. This incidentally helps to dissolve and loosen the false membranes, so far as it can reach them. Calomel, 10 grains, iron chloride, 1 dram, and sulphate of quinia, 1 dram, may be given in honey smeared on the back teeth, to be swallowed at leisure. The throat may be wrapped in damp cloths, wrung out of hot water and covered with dry, or it may be enveloped in a strong mustard poultice for at least an hour.

Inhalations of vaporized (heated) ammonium chloride, or of the fumes of burning sulphur suitably diluted with air and water vapor, or of eucalyptol, or even of burning tar will often prove useful.

In cases threatening asphyxia tracheotomy must be the instant resort. After the operation the relief from dyspnoea and the better aeration of the blood often determine marked improvement, and the diseased laryngeal mucosa can be easily reached and dressed with antiseptics carried on a sponge fixed on the end of a staff.

PHARYNGEAL, AND LARYNGEAL POLYPI.

Pediculated tumors. Dyspnoea through change of position, operation by ecraseur, snare or cricoid incision.

Tumors of varied structure developing in or beneath the mucosa of pharynx or larynx often become slowly detached until they hang by a loose pedicle, and having much latitude of movement they may at times slip between the arytenoid cartilages or into the glottis producing the most urgent or even fatal dyspnoea. Pediculated tumors in the posterior nares lead to the same accident. In one case of multiple small tumors on the pharyngeal mucosa of the horse, the largest and loosest, attached to the front of the epiglottis, was occasionally displaced into the larynx threatening instant asphyxia. One such attack supervened on the opening of a suppurating guttural pouch by the writer, necessitating prompt tracheotomy. A time was set for the removal of the polypus, but the tracheotomy tube having been accidentally displaced during the preceding night the patient died of suffocation. Dick mentions a polypus growing from the interior of the larynx and causing loud *roaring*. Such tumors may be removed by operating with the *ecraseur* through the mouth, or by a snare passed through a long narrow tube and used to seize and twist through the pedicle. If the polypus grows from the laryngeal walls, it may be best reached by incision through the cricoid cartilage and crico-thyroid membrane as in the operation for *roaring*.

DYSPNŒA LARYNGEA. ROARING. HEMIPLEGIA LARYNGEA.

Generic name for common symptoms. Low and high notes. Grunter, roarer, whistler, piper, highblower. Pace or effort develops. Causes : of temporary roaring, inflammations, abscess, phlebitis, choking, dropsy, petechial fever, neoplasms or phlegmons along vagus. Causes of inveterate roaring, paresis of left recurrent laryngeal nerve, fatty degeneration of left arytenoid muscles, fracture of facial bones, polypi in air passages, chronic thickening of mucosa, foreign bodies in passages, tumors of lymph glands, abscess of guttural pouches, pseudo-membrane, laryngeal ulceration, ossification, distortion, fracture of cartilages, action of forcible inspiration, leading on left side, deep origin of recurrent nerve, effect of chest disease, and violent heart action, examples of morbid conditions impairing innervation. Lesions in muscles, and nerves. Facial palsy, poison (chick vetch, winter vetch, lead, fungi, moulds). Intermittent roaring. Hereditary roaring. Symptoms, grunting when coughed or threatened, heavy draft, galop, noise, laryngeal tremor, cold as a complication, roaring with expiration, lesions. Treatment, its use. Prevention, avoid breeding roarers, bearing reins, chick vetch, lead. Tonic medication, caustic to mucosa, firing, setons, iodine, pad nostrils, tracheotomy, arytenectomy, electricity.

This is the name of a symptom rather than a disease. It implies a sound made in breathing in connection with some contraction of the air passages. The term is however usually reserved for those conditions in which the sound results, from chronic disease or malformation, the noise attendant on laryngitis and other acute diseases being rarely spoken of as *roaring*. In neither case does the noise indicate more than that there is some impediment to the ingress and egress of air through larynx or trachea.

The pitch of the note varies exceedingly with the causes that produce it and with the hurried nature of the breathing. There have thus arisen the epithets of *grunter*, *roarer*, *whistler*, *highblower*, *piper*, *trumpeter*, *wheezier*, etc. The most common distinctions are those of *roaring* and *whistling*. The *roarer* produces a loud deep bass sound in inspiration, the larynx or windpipe being only slightly narrowed while the *whistler* or *piper* produces a shrill blowing or sibilant noise because of the greater constriction of the passage. The term *grunter* is derived from the facts that a *roarer* usually makes a grunting noise when struck or threatened with a cane, and that when the upper cartilages of the windpipe are pinched between the finger and the thumb the

resulting cough is prolonged and sometimes like a *grunt*. A *wheezer* is usually suffering from spasmodic contraction of the bronchial tubes, from broken wind (heaves), or from chronic bronchitis. A *highblower* should never be spoken of in the same connection, as the noise is made from a playful flapping of the false nostrils or soft palate and disappears when the animal is put to the top of his speed. It is from confounding *highblowing* with *roaring* that *Eclipse* and other brilliant performers on the English turf have been erroneously pronounced *roarers*.

The noise produced by the *roarer* is not heard while he stands quiet, nor in many cases even during a short trot or gallop. Such horses are in consequence often sold at the hammer and the purchaser is grievously disappointed to find that what he thought a sound horse is absolutely useless for the purpose for which he designed it.

Causes. Before noticing the symptoms of roaring a consideration of the causes will be useful to enable the reader the better to understand the signs by which the different forms are manifested.

Causes of temporary roaring. Whatever impedes the current of air causes roaring. Hence inflammatory diseases of the nose, throat, windpipe or bronchial tubes; abscess of one or the other of these parts; inflammation of a jugular vein and pressure on the trachea or vagus nerve by the resulting swelling; choking; the swelling in the neck consequent on the cutting open of the gullet for the relief of choking; thickening of the nostrils from dropsy; loss of a jugular vein; purpura hemorrhagica, etc.; and swellings pressing on the vagus nerve, and which may be situated at the base of the brain, in the neck or in the anterior part of the chest. Also temporary infiltration of the laryngeal mucosa.

Causes of inveterate roaring. The one great cause of roaring and that which sustains the disease in nineteen cases out of every twenty is paralysis of the left *recurrent nerve* of the larynx and wasting of several of the arytenoid muscles on that side. It may be well, however, first to notice the less frequent causes and wind up with this more common one. 1st, Fracture with distortion of the nasal bones and narrowing of the nasal passages (Gamgee). 2nd, Polypi and other tumors of the nose, pharynx, larynx, windpipe or bronchi. 3rd, Chronic thickening of the

nasal mucous membrane, the result of inflammation. 4th, The presence of foreign bodies in the nose, as for example balls coughed up from the pharynx. 5th, Hering records a case resulting from the closure of the posterior opening of one nasal chamber by a membrane. 6th, Cancerous or melanotic deposits in the lymphatic glands above and to each side of the pharynx and larynx. 7th, Distension of the guttural pouches by inspissated pus. 8th, Chronic thickening of the mucous membrane of the larynx consequent on inflammation. 9th, The formation of a projecting fold of the mucous membrane or of a new production (false membrane) in the windpipe as the result of inflammation. Such false membranes have been known to become detached at their median part and remain attached at their two extremities thus constituting a band stretching from one side of the windpipe to the other. 10th, Ulceration of the membrane of the larynx particularly on the projecting folds circumscribing the glottis. 11th, Ossification of the laryngeal cartilages and loss of their elasticity. 12th, Distortion of the cartilages of the larynx, most commonly from unduly tight reining and pulling the nose in toward the chest. In such cases the cartilages of the larynx and those of the windpipe adjoining being compressed slide within each other, and the enclosed edge projecting within the air tube materially diminishes its calibre. 13th, Fracture of one or more rings of the trachea. This usually results from blows, as in running the neck against the back of a cart or wagon. The cartilaginous rings are usually broken at their median part in front and being retained together by the investing elastic tissue which enables the pieces to move on each other as by a hinge, and being approximated by the contraction of the trachealis muscle above, the ring is flattened from side to side and the channel for the passage of air correspondingly decreased. This flattening can be easily felt by the hand, in the living horse. 14th, A peculiar congenital distortion of the trachea caused by the curling in of one end of each cartilage of the windpipe and the straightening out of the other. This occasionally proceeds so far that the gullet is lodged in the interspace overlapped and hidden by the free ends of the cartilages, the diameter of the windpipe being proportionately diminished. Distortions and fractures are usually overlooked by veterinarians but from the frequency with which the author has

met with them in his dissection he is convinced that they deserve greater attention than has been accorded them. 15th, Percivall records a case of inveterate *roaring* in which even tracheotomy having failed to cure, the patient was destroyed and the lungs found to be extensively consolidated, many of the air tubes having been so compressed as to be almost impervious. I have known a case of roaring from the presence of a pedunculated tumor in the lower end of the windpipe where it divides to enter the lungs, and the same result may ensue from the partial obstruction of the bronchial tubes by masses of tenacious mucus in chronic bronchitis. 16th, Rupprecht relates a case caused by depression of the cartilages on the left side of the larynx and twisting of the left arytenoid cartilage, the point of which was turned outward so that the upper opening of the larynx was actually widened, but the lower border of the arytenoid left free, naturally slid inward toward the centre of the lumen at the level of the vocal cords, and thus caused the stertor. 17th, Similarly Bouley, Jr., notes a case of false joint in the middle of the arytenoid cartilage, so that the detached posterior portion, was drawn into the median line by the combined action of the arytenoideus, and crico-arytenoideus posticus muscles. In this case the passage for the air was almost entirely closed. 18th, Other causes are ossification ankylosis of the laryngeal cartilages, chondritis of arytenoid or epiglottis with formation of an enchondroma, fracture of a cartilage with a similar cartilaginous growth, the growth projecting into the lumen, and small cartilaginous vegetations on the vocal cords. Dupuy records the presence of a mass of erectile tissue in the arytenoid mucosa, and Cadeac hypertrophy with contraction of the connective tissue which binds the two arytenoids together. 19th, The immediate cause of *roaring* in the immense majority of cases is the paralysis and fatty degeneration of certain muscles on the left side of the larynx. The muscles supplied with motive power by the left recurrent laryngeal nerve (Crico-arytenoideus posticus, Crico-arytenoideus lateralis, thyro-arytenoideus, and the left half of the arytenoideus) are those constantly and exclusively affected, while those supplied by the *superior laryngeal nerve* and first cervical (Crico-thyroideus, hyo-thyroideus and hyo-epiglottideus) remain unchanged. The left recurrent nerve is also wasted and

considerably attenuated as compared with that on the opposite side. The *modus operandi* of this paralysis and wasting in the production of *roaring* is beautifully seen when the upper part of the windpipe is laid open so as to expose the interior of the larynx in laryngectomy. The triangular opening of the glottis is seen fairly dilated while the muscles are relaxed in the act of expiration. As soon, however, as inspiration commences the left arytenoid cartilage slides completely into the passage, its lower border projecting so much to the right that it forms a prominent crest extending beyond the median line and in some cases closely approaching the right wall of the larynx, the superior elastic and free border of the same cartilage meanwhile gets drawn inward by the suction power of the air so as to block up the passage still more. The closure of the glottis being thus seen to be largely controlled by the current of inspired air, it becomes evident that any increase in the force of the current will aggravate it and a decrease will lessen the extent of the closing and alleviate the distress of breathing. This fact furnishes a means of palliating the symptoms. (See treatment.) It explains moreover why roaring should not be heard in quiet breathing and why it should increase in force and in pitch as the respiration becomes more and more hurried. It further accounts for the noise being heard only during the act of inspiration, the outward rush of the air in the expiratory act being of itself sufficient to carry this valvular cartilage out of the passage and secure a free and unimpeded current.

The paralysis and wasting of the left recurrent nerve and muscles are in their turn due to very varied morbid states.

It may commence in the larynx from distortion of its cartilages and inflammation of the mucosa, in which case the wasting of the nerve is probably a result of its prolonged inactivity. This mode of origin is strongly insisted on by Percivall, and no doubt occasionally arises. Under this explanation, however, it is difficult satisfactorily to account for its almost invariable occurrence on the left side. The mere fact that the horse is habitually approached on this side and more commonly turned toward it is a most insufficient reason.

Even if admitted it utterly fails to explain the immunity of muscles supplied by the superior laryngeal nerve.

The fact that a horse has usually a hard and soft side of the mouth and carries the head slightly to the latter is no better explanation, the tender side is not always the left.

More commonly the disease arises at some other point near the origin or in the course of the nerve, and the changes in the larynx follow as the consequence of deficiency or entire absence of motor innervation. Many cases can be cited in which such an origin was unquestionable, and on the hypothesis that this is the true and constant history of the development of the malady, its regular occurrence in the left side and the absence of all signs of wasting in the muscles supplied by the superior laryngeal nerve are alike perfectly explainable.

Let it be noted that the vagus nerves (right and left), of which the recurrent laryngeal are branches, originate from the base of the brain, pass down the neck on the deep aspect of the jugular vein in company with the carotid artery; that on entering the chest the right vagus nerve gives off its recurrent branch which proceeds at once up the neck along the course of its parent trunk till it reaches the larynx, to the muscles on the right side of which it is distributed; that the left vagus nerve on the other hand proceeds backward in company with the left innominate artery as far as the base of the heart, where on about the level of the space between the sixth and seventh ribs it gives off the left recurrent nerve; that this left recurrent nerve closely applied at its point of origin to the great parent arteries turns round the posterior aorta enclosing it in a loop, and gaining the lower end of the windpipe follows its course to the larynx. It will thus be understood how many chest diseases may implicate the left recurrent nerve, and from which the right, which extends no deeper than between the two first ribs may be completely exempt. The frequent supervention of roaring as a sequel of chest diseases receives in this an ample explanation. Its connection with pleurisy becomes especially probable, as the nerve lies in contact with the surface of the pleura alike in its descending and ascending course within the chest.

Finally the loop encircling the posterior aorta exposes the nerve to constant stretching and shocks from the heart's action during violent exertions and in excited states of the circulation generally. Vaerst and Sussdorf show that the nerve is habitually flattened

between the posterior aorta and trachea, the effect being worst when the heart's action is excited.

The relatively great prevalence of *roaring* in the thoroughbred English race horse at home (5% in the army in 1888) and in the descendants of this race in other countries, implies a special cause, connected with conformation or usage. The spare habit of the body, the lack of fat, and the violent exertion demanded of the animal have been adduced in explanation. The last named is commonly the most potent. A race of horses with great energy and extraordinary speed is of necessity characterized by a large cardiac development, and with steady and exacting training this is increased to meet the demand. The more exacting the training, if kept short of exhaustion, and if successful in attaining the highest possibilities of the animal, the greater the increase of the heart. The larger and more powerful the heart the greater its impulse with each contraction and the greater the liability to injury of the nerve: 1st, by the concussion of the heart itself; and 2d, by the compression of the nerve between the posterior aorta on the one hand and the trachea or pulmonary artery on the other. The same cause operates in cases in dogs used in draught (Müller). Cases of laryngeal paralysis in man in connection with hypertrophy of the right heart, are recorded.

Horses suffer more numerously than mares especially in breeding districts where the mares are rested during pregnancy and nursing.

A moderate wasting of the arytenoid muscles does not necessarily cause roaring, especially in animals that are not subjected to trials of speed or other violent exertion. Considerable atrophy of these muscles is often found in the subjects of dissection in which the infirmity had not been suspected during life. This adds especial emphasis to the unwontedly violent contractions of the heart as a cause, whether this depends on pericardial, pleuritic or pulmonary inflammation on the one hand, or on extreme hard work on the other. This also serves to explain the comparative absence of the symptom from cattle and dogs, that are not compelled to severe exertion, since on the supervention of dyspnoea, such animals instinctively slacken their pace and thus anticipate or arrest the roaring. Müller's *roaring* dogs were used in draught and thus compelled to severe exertion irrespective of their own desires.

It remains to notice a few instances in which dissection established the connection of interference with the nerve at some part of its course and the existence of roaring.

(a.) Godine found in a roarer a tumor about the size of a chicken's egg, pressing on the commencement of the pulmonary artery. He attributed the roaring to the impaired circulation of blood in the lungs by the pressure on the artery. Considering that the tumor must have been precisely in the situation of the left recurrent nerve at its point of origin, it becomes much more probable that the symptom resulted from pressure on this nerve.

(b.) The elder Bouley found in one case a considerable engorgement of the group of lymphatic glands in the anterior part of the chest and through the center of which the left recurrent nerve passed. Cary notes sarcoma of the posterior cervical and prepectoral glands.

(c.) Fergusson of Dublin dissected a roarer in which he found besides some tumors of the lymphatic glands in the pelvic and sub-lumbar regions, an indurated and enlarged gland about four inches behind the anterior opening of the thorax. The recurrent nerve between this and the larynx was wasted so that its fibres could scarcely be recognized, the laryngeal muscles on that side were atrophied, and degenerated, and the glottis distorted and partly closed. Fergusson has in his description made the mistake of writing the *right* for the *left*; it is evident that the *right recurrent nerve* could not possibly pass through a tumor in the situation described.

(d.) Gamgee furnishes a drawing of an immense tumor filling up the anterior part of the chest, pressing on the vagi and recurrent nerves and causing roaring. Such tumors and inflammatory engorgements have been frequently noted since, and may be of the most varied kind—strangles, lymphadenoma, melanoma (in gray or white horses), tubercle, sarcoma (Cary), carcinoma, etc. Phlegmonous inflammations of the bronchial or mediastinal glands, aneurism of the aorta, sacculated and impacted gullet, are other causes of compression and wasting of the nerve.

(e.) The Clinique of the Alfort Veterinary School furnishes the following among other cases of roaring consequent on inflammation of the jugular vein. A well-bred and very fast English thoroughbred had been used for two years by his owner who was

a hard rider. In June, 1857, he was bled as a preventive (saignée de precaution), suppurating phlebitis was induced and was only cured at the end of six weeks. When again put to work he proved a *roarer* and was still affected when seen six months later.

In this relation it may be noted that the swelling in connection with the inflammation of the vein extends easily to the subjacent vagus and recurrent nerves, leading to their inflammation, functional inactivity and atrophy. Bleeding is usually done on the left side of the neck so that paralysis and wasting would still be on the same side. Happily with a more humane system of treatment, accidents of this kind are less frequent than formerly. Glöckner furnishes a case which followed thrombosis of the carotid.

(f.) Reynal reports several cases in which *roaring* had occurred as a sequel of inflammations and abscess about the throat, and in which infiltrations or gray or yellow indurations had taken place in the areolar tissue around the vagus nerve. As nothing is more common than to find *roaring* resulting from severe sore throat, parotitis, etc., this may explain its occurrence.

Mandl first carefully examined the paralyzed muscles which present to the naked eye a flattened and wasted appearance in marked contrast to the full well-rounded forms of those on the opposite side. They differ no less in color. In place of the deep red of the healthy muscles those on the diseased side are of a yellowish white hue with here and there a pink streak indicating the position of some unchanged muscular fibre. When placed under the microscope the healthy elements of the muscular fibres (sarcous elements) are seen to be replaced by granules of fat. The nerve (recurrent) is not only visibly wasted, but its tubular white substance (white substance of Schwann) can no longer be recognized and it approximates closely in character to a filament of ordinary white fibrous tissue.

20th. Muscular paralysis due to other causes and even located in different parts has been known to give rise to *roaring*.

Goubaux and others have noted the occurrence of roaring from paralysis of one nostril, alike when the loss of power was special to the nasal muscles or common to all on one side of the face. This, however, is snuffling rather than laryngeal stridor.

21st. Roaring apparently from paralysis of the laryngeal — muscles has been seen frequently in animals fed on the seeds of leguminous plants and specially of the *Lathyrus Cicera* (*Lathyrus Sativus*, *Stendel*.) The whole family of the *Leguminosæ* is open to suspicion as occasionally containing a poisonous principle capable of inducing paralysis in animals fed on them. The *Lathyrus Sativus* induces paralysis in man and the domestic animals in some parts of India (Sleeman, Irving). The common cultivated tare (*Vicia Sativa*) is well known to induce general paralysis, commencing with the hind extremities, when fed to horses at the period of ripening in Great Britain. In France the chick vetch (*Lathyrus Cicera* or *Sativa*) has been repeatedly noticed to lead to the development of roaring apparently from paralysis of the laryngeal muscles. Horses fed on 17 lbs. daily (straw and seeds) were attacked with roaring in five days. They gained in flesh and vigor, had a smooth shining coat and supple skin, and standing at rest presented nothing amiss, but after ten minutes trot they were seized with roaring and if not stopped they soon fell to the ground, with symptoms of impending suffocation (Delafond). Horses fed heavily on the winter vetch with cut hay and molasses were attacked with roaring if gently exercised for one or two minutes. It came on suddenly and threatened instant suffocation. One horse fell and lay half an hour in a frightful state of dyspnœa. More commonly they recovered after a few minutes rest. In the interval no disturbance of breathing nor any change of appetite attested the slightest deviation from health. Reynal, Cruzel, Caffin, Motte and Ayrault mention similar occurrences.

22nd. The paralysis of chronic lead poisoning will also cause roaring.

Occasional or intermittent roaring. Puzzling cases are met with in which a horse will *roar* at one time and not at another. In such cases the veterinary profession has incurred an amount of odium which was by no means deserved. Two veterinarians, equally respectable and talented, appearing in a Court of Justice to swear to the same animal which they had examined on different days, respectively pronounce it a roarer or a sound horse, as it happened to be at the time of the respective examinations. Such cases have been differently accounted for.

Slight colds or *sore throats* may cause roaring so long as they persist. Tight reining with the nose drawn in toward the chest induces a stridor in certain animals by distorting the larynx and trachea. Some horses with thick necks, badly set on heads, and in a state of obesity, roar, yet the symptoms subside when the superfluous fat is got rid of and they are brought into hard working condition. Stallions are very liable to make a noise from this cause. In a case of roaring which disappeared when the horse had been exercised for some time Leblanc diagnosed an œdema of the glottis which was absorbed under the increased movement of the parts. He did not test his opinion by dissection.

Roaring sometimes hereditary. That roaring runs in families there can be no doubt, but the direct cause appears to be mostly the transmission of a faulty conformation. A head with faulty shape and badly set on; a thick, short neck, deficient in mobility, or a small, narrow chest, predisposed to acute diseases, descends from parents to offspring, entailing a predisposition to roaring. The large Normandy horse is notoriously subject to roaring, but then he is equally characterized by a big, coarse head, narrow forehead and nostrils, big jowl, and narrow intermaxillary space. In all breeds this form is very subject to roaring, because of the stiffness of the neck and tendency to compression of the larynx. With the head badly set on, as it is almost of necessity in these animals, everything is done to produce roaring. Not only is the head cruelly reined in at work, but the horse is kept a great part of his time in the stable in the same or even in a worse condition, the larynx meanwhile unnaturally compressed between his narrow jaws, and the nerve compressed or the larynx distorted.

It must be added, however, that like some other acquired distortions or alterations roaring may repeat itself in the progeny. Goodwin mentions an instance of it on the female side through three successive generations of thoroughbreds. Of transmission on the side of the male the following instance is noteworthy: M. Liphaert, an extensive proprietor in Livonia, bought a first-class English thoroughbred stallion. His progeny were healthy until he became a roarer at ten years old. All his foals, got after this date, followed the sire in becoming roarers, and, it is important to observe, almost all at the age of ten years.

Symptoms. These, of course, are manifest enough while the animal is sufficiently excited to give rise to the noise. Certain indications may be obtained even while the animal stands in the stable. If *cough* is excited by pinching the upper rings of the windpipe it is prolonged into a groan. If led up to face a wall and suddenly threatened with a cane the abrupt inspiration which results is attended by a *grunt*. The absence of these symptoms is not, however, sufficient to establish the non-existence of roaring. The horse must be galloped or put to heavy draught to fully test the breathing organs. Galloping up a steep hill is perhaps the best test. A gallop over a recently ploughed field is about equally good. Soft pasture land or an unpaved road is preferable to Macadam or pavement. Galloping in a riding school on the soft tan is an excellent measure as the sound is confined and the animal is always within earshot of the examiner. The person examining should either ride the horse himself or have a disinterested party, in no way connected with either buyer or seller, to mount him. If the rider is in the interest of the seller he may contrive to slacken the pace before he reaches the examiner, or by irritating the horse may make it difficult to approach him immediately on his being pulled up. If in the interest of the buyer he may succeed, by the use of a powerful bit, in drawing the horse's nose in to the chest, or by compressing the larynx with a tight throat latch he may produce noise in breathing when the animal is suddenly brought to a stand. Unless the course is up a steep hill or over a ploughed field the horse should be galloped from five to ten minutes; he should be then made to pass close to the examiner at full speed, and finally brought up suddenly by his side and without any previous slackening of his pace. The ear should be at once placed close to the nostrils, when the slightest abnormal sound accompanying the inspiratory act will be at once recognized.

Draught horses are sufficiently tested by driving them in a heavy vehicle or one with the wheels dragged. By walking alongside or keeping the ear near to the nostrils any harsh sound additional to the normal blowing noise of hurried breathing is easily noted.

The finger placed on the larynx detects the strong vibratory tremor, and Friedberger notes that the left arytenoid is much more easily displaced than the right, increasing the stridor.

If the horse is, at the time of examination, the subject of a cold, sore throat, or other acute disease of the air passages no importance is to be attached to any noise made in breathing, but he cannot be pronounced a *sound* horse until, this malady having passed off, it is found on careful examination that no such sequel has been left.

Among the most puzzling cases are those in which the roaring occurs with periods of intermission. If the horse has for a short time eaten vetches or ripe rye grass or has taken lead in water or food this may account for its temporary access, and unless the same is again allowed a recurrence is not to be looked for. If due to the occasional displacement of a pedunculated tumor of the nose or pharynx and its interference with the action of the larynx its existence may be recognized by careful examination, diminished current of air through one nostril, etc. But there remain some *rare* cases in which there are no such appreciable causes, and yet the horse would be pronounced *sound* or *unsound* as examined at certain intervals. On this subject more information is desirable.

The following varieties of roaring will be distinguished from that of paralysis by the occurrence of the sound in both acts of breathing (expiration and inspiration):—distortions, tumors or foreign bodies in the nose: tumors about the throat, in the windpipe or bronchi:—distortion of the windpipe, from tight reining, fracture or congenital deformity:—and the presence of a false membrane stretching across the windpipe.

Examination by manipulation, auscultation and percussion along the whole length of the air passages alike during rest and after exercise, may enable one in unusual cases to recognize the structural changes that give rise to roaring.

Treatment. This was long considered as hopeless, yet preservative and palliative measures are usually accessible, whilst even cures can be effected in certain conditions.

Preventive treatment. First may be noticed the rejection for breeding purposes of all animals possessing those conformations of head, neck and chest already referred to as conducing to disease of the air passages or distortion of the larynx or windpipe. Equally ought all roarers to be set aside unless the exciting cause is accidental such as fractures of the nasal bones, of the trachea, the existence of polypi, etc. Stallions that make a harsh noise

in breathing from an accumulation of fat about the throat are not necessarily objectionable.

The employment of the check rein so as to compress and distort the larynx is to be avoided. If bearing reins are used in horses having short thick necks and badly set on heads and especially with intermaxillary narrowness they should be passed through rings in the cheek piece of the bridle or between the ears and over the forehead (overdraw check) so that while the head is elevated the nose may be projected forward after the Russian fashion of equitation. This measure has indeed appeared to cure several cases of roaring. I have met with fewer roarers in the same number of horses in America than in England, and this I attribute in part to the better mode of using the bearing rein on this side of the Atlantic.

The Chick Vetch (*Lathyrus Cicera*) should be excluded from the fodder of horses or used in small proportion only. In man it is found to be injurious when it forms a twelfth part of the bread used and gives rise to paralysis if it amounts to a third (Aitken).

Paliative and Curative treatment. Medical treatment will prove useless in the great majority of cases: as for example in paralysis and degeneration of the muscles, in ossifications, fractures, or distortions of the cartilages, etc., etc. Nevertheless where there is merely thickening of the membrane of the larynx alterative and tonic treatment may be successful especially if associated with iodine ointment or active blisters applied to the throat. A case is reported by Dupuy which a course of arsenic cured. In these cases as well as in those due to ulceration of the membrane the application of caustic by means of a staff and sponge as advised in laryngitis may prove beneficial. In some cases of this kind the application of the firing iron to the region of the larynx has an excellent effect. Setons have proved useful in some cases.

In cases due to tumors or enlarged glands pressing on the air passages the internal use of iodine and other alteratives and diuretics, and the local applications of iodine or mercurial ointments or of blisters have been successful. Failing in this the tumors may be removed with the knife when accessible.

If by auscultation the existence and position of a band of lymph can be made out, tracheotomy may be performed and the band

excised. Percivall with reason doubts the possibility of the diagnosis.

In cases due to distortion of the larynx from tight reining the bearing reins should be dispensed with or rearranged so as to encourage protrusion of the nose, and the horse should be bitted to the side chains or straps in the stall several hours daily so that the head shall be elevated and the nose protruded.

When *roaring* depends on paralysis of the laryngeal muscles, a mode of palliation may be adopted as practised by the London omnibus and cab men. A strap is fixed round the nose supported by a strap passing down the middle of the face and the cheek piece of the bridle on each side and buckled beneath the chin. On the inner side of this strap where it passes over the false nostrils, is attached on each side a semiovoid pad which presses on the flap of the nostril and regulates the entrance of air. The principle on which it acts will be understood when we consider that the paralyzed cartilage is drawn into the passage by the rush of air and that the closure of the channel is more complete and the roaring more marked in proportion to the force of the current. The pads by lessening and regulating the rush of air into the lungs thus leave the passage in reality more open and largely obviate the difficulty of breathing and the noise.

In extreme cases with the structural lesion in the head, throat, or upper two-thirds of the neck relief may be secured by tracheotomy.

A more radical operation is that introduced by Günther for the excision of the left arytenoid cartilage. As improved by Möller and others this consists in an incision through the cricoid cartilage and crico-thyroid membrane (even the first rings of the trachea) and the complete extirpation of the left arytenoid cartilage and left vocal cord. The manipulations belong to surgery. The result is satisfactory in removing the violent dyspnoea in hurried breathing and in very favorable cases in obviating noise altogether though not permanently. More commonly some stridor remains but not enough to interfere with pace or with heavy draft.

The other operations:—excision of the left vocal cord and ablation of the ventricle (Günther);—the fixation of the arytenoid to the thyroid (Günther);—the excision of the left vocal

cord and suture of the crico-arytenoid membrane to the thyroid (Merilat);—the suture of the crico-arytenoid membrane by incorrodible gold wire to the thyroid (Williams);—and the suture of the left recurrent laryngeal nerve to the parent trunk of the vagus near the larynx; have proved successful in suitable cases.

The best and safest resort is the removal of the left vocal cord and of the entire mucous lining of the ventricle of the larynx. (W. L. Williams). Prepare the subject by performing tracheotomy in the upper third of the neck. A plug may be placed in the windpipe above the tracheotomy tube and stitched to the skin. This will prevent the descent of blood into the lungs during the operation. Then the larynx is opened in the median line, the incision being carried through the crico-thyroid ligament, the cricoid cartilage and the crico-tracheal ligament. It is sometimes imperative, though otherwise undesirable, to carry this down through the first two tracheal rings as well, then with the head fully extended and the laryngeal wound held widely open by retractors, the left vocal cord and the mucosa covering its inner surface and closing in the ventricle, is seized with the tenaculum forceps and carefully dissected off by the aid of scissors, scalpel or razor-like arytenectomy knife having a long handle. The incision is commenced on the tracheal aspect of the vocal cord, down towards its base, and must stop short of the ventricular mucosa which is held stretched until it has been detached from the entire ventricular cavity, when it is cut loose from the laryngeal mucosa above and removed. Should it become accidentally torn it must be seized anew by tenaculum or dressing forceps and every portion carefully dissected off. Clots must be removed or pushed into the pharynx to be swallowed, bleeding points stopped by pressure or torsion, and the surface disinfected and covered with a mixture of iodoform and tannin, or better with aristol to which a slight addition of adrenalin has been made. In case of free and persistent oozing plug the larynx with a roll made of a single strip of gauze, medicated with one of these dressings, suture it to the skin and leave it in place for twenty-four hours. Dress the wound daily, or as may be wanted, by means of pledgets of medicated cotton safely held in long dressing forceps. For the first eight or ten days feed on milk only, given in a bucket set on the ground.

On the tenth day examine the interior of the larynx in a good light and excise or cauterize any exuberant granulations and repeat this if necessary. The tracheotomy tube may now be removed, but reinserted if there is still much difficulty of breathing.

Cary omits the tracheotomy when possible, restricts the incision to the crico-thyroid membrane, excises both right and left vocal cords, and dissects off the left ventricular mucosa and an elliptical vertical strip of the mucosa behind and in front of the ends of the vocal cord. This may be an inch long by half an inch wide in the case of the posterior one.

This operation has the advantage of avoiding any wound of cartilage, apart from the cricoid, and the formation of exuberant granulations which are so common on cartilaginous wounds. It further secures the formation, in the seat of the former ventricle, of a dense cicatrix which permanently binds the arytenoid to the inner side of the thyroid cartilage, thereby preventing its displacement into the lumen so as to shut off the passage of the air.

Another obvious line of treatment is by the use of electricity locally and of strychnine internally. A weak current kept up for fifteen minutes may be sent from the positive pole in the left jugular furrow to the negative pole over the left side of the larynx. Strychnia in the dose of two grains may be given daily in the food, or in half that amount hypodermically over the left side of the larynx. This would be useful only in the early stages with little or no fatty degeneration of the muscles. It is the recent, slight cases that are benefited in this way, and some such have even recovered under light firing over the region of the larynx.

ŒDEMA GLOTTIDIS.

Diseases complicated by this. Seat. Abundance. Duration. Sequels. Symptoms: sudden dyspnoea, swelling of throat, pits on pressure, differentiation from croup. Treatment: cold, ice, astringents, scarification, tracheotomy.

This is usually a complication of acute laryngitis, but it may be a manifestation of other forms of local disease—tuberculosis, glanders, purpura hæmorrhagica, pseudo-membranous inflammation—or it may be a result of a more distant affection, like disease of the heart, lungs, or kidneys. It has also followed on wounds of the soft palate; on injuries of the palate or tonsils by the rasp used on the molar teeth; on strangulation (agony); on phlebitis of the jugular with paralysis of the sympathetic nerve or obstruction to the return of venous blood from the larynx; and on tumors of various kinds in the vicinity. It has been attributed to an excessive dose of potassium iodide, and any violent irritant applied to the laryngeal mucosa may induce it. As a complication of local inflammation it consists in an excessive serous exudation into the submucosa, around the base of the epiglottis and extending to the whole larynx and pharynx. It may thicken the parts by half an inch, causing complete closure of the glottis. In favorable cases it may subside as rapidly as it rose, while in others it may result in ulceration or abscess. The infiltration has usually a clear watery aspect, but is sometimes a dull red. When incised an abundance of serum escapes mixed, in certain cases, with pus.

Symptoms. In the course of one of the above named affections there comes on suddenly extreme dyspnoea, with stertorous breathing, a suffocating cough, and intense anxiety. The stridor is first with inspiration alone and later with expiration as well. The eyes are bloodshot and protruding, the pulse small and rapid, movements uncertain, and the skin moist with sweat. There is manifest swelling of the throat and manipulation leaves the imprint of the finger.

When symptomatic of some distant affection it is at once slower in its result and more persistent.

The local pasty swelling and the absence of any false membrane suffice usually to distinguish it from croup which it so

closely resembles in the suddenness of its onset, and the violence of its manifestations.

The less urgent cases may be treated by application of cold water or ice to the throat, and the injection of solution of chloride of iron or alum into the fauces. Or the throat may be painted with tincture of iodine and rubbed with the palm to favor distribution and absorption of the exudate. In dogs the mouth may be opened widely and the dropsical membrane pricked at intervals to drain off the liquid. In the most acute cases the prompt adoption of tracheotomy is the only means of saving life.

LARYNGEAL, HYPERÆSTHESIA. CONVULSIVE COUGH.

Convulsive cough with visible lesions—without. Excitants : cold air, or water, rough or dusty food, irritant agents inhaled or swallowed. Treatment : hygienic, nerve sedative, expectorant, tonics, muriate of ammonia, sulphur dioxide, silver solution, ferric chloride, alum, derivatives, elimination, aromatic, dietetic.

The chronic or paroxysmal cough may often be traced to the presence of tumor, ulcer, local inflammation, or parasite, but in some instances no local trouble is recognizable, the general health remains good, and yet the throat is abnormally sensitive and a cough or fit of coughing may be roused by passing into the cold air from a warm stable, by cold water in drinking, by inhaling irritant gas, by the passage of rough or fibrous food, or by handling the larynx. There is undoubtedly a hyperæsthesia of the larynx, and the horse and dog being more exposed to severe demands on the physical and nervous systems, are especially liable to suffer.

Treatment must be adapted to the conditions. Over-work, damp, unhealthy buildings, and all appreciable health depressing causes must be corrected, and a course of iron and nux vomica with moderate open-air exercise may be tried. Borax, bromide of potassium, and extract of hyoscyamus, made into an electuary with molasses or honey may be smeared upon the molars four or five times a day. In obstinate cases the inhalation of the fumes of burning sal ammoniac or sulphur, or the direct application to the larynx of dilute solutions of silver nitrate, ferric chloride, or alum may benefit. The throat may be blistered by cantharides or mustard. Care should be taken to keep the functions of bowels and kidneys normally active, to protect the patient against cold and damp, and to give nutritive but non-stimulating and easily digested food, as for the horse, bran mashes, roots, grass or scalded hay, and for the dog pulped flesh, soup and mush. Sometimes benefit can be obtained from the vegetable aromatics and stimulants as eucalyptol, tar, turpentine, balsams of Tolu and Peru, tincture of anise, fennel, etc.

INFECTIOUS DISEASES OF THE THROAT.

Infections are in many respects the most serious affections of this region, but their consideration must be sought under strangles, distemper, diphtheria, anthrax, actinomycosis, tuberculosis, glanders, etc. (Vol. IV). *Aspergillus* (Vol. V).

PARASITES OF THE THROAT.

Leeches. These taken in with the water will sometimes fasten themselves on the walls of the pharynx or even on the lips of the larynx, producing cough, sore throat, difficulty of swallowing, bleeding from the nose (or mouth) or dyspnœa. They are to be removed as recommended under parasites of the nasal chambers. (Vol. V.)

Æstrus Larva. Bots. In horses and mules the larva of the æstrus sometimes attaches itself to the mucous membrane of the pharynx or even of the larynx producing chronic irritation, cough and even dyspnœa. A chronic sore throat with nasal discharge, occurring in autumn or winter, in the absence of fever or constitutional disorder may be found to depend on these parasites and to recover when these have been removed by the hand. (Vol. V.) *Linguatula Tænioides.* (Vol. V.)

GUTTUROMYCOSIS OF SOLIPEDS.

Aspergillus. Complications : ulceration, lesions of adjoining parts, food in lungs, hepatization, gangrene. Treatment, by incision, sulphur dioxide, iodine.

Rivolta and Bassi have found in the guttural pouches of horses and a mule, an advancing ulceration of the mucosa partially covered with crusts composed largely of the mycelium, conidia and spores of *Aspergillus* or a closely allied fungus. In the mule the ulcer had opened into the carotid artery causing a profuse epistaxis. In the three horses there was dysphagia, and the food, descending to the lungs, had caused pulmonary hepatization and gangrene. The description of the ulcers lead Raillet to infer the existence of glanders and that the presence of the *aspergillus* was accidental, rather than a causative factor. In parallel cases the opening of the guttural pouch and injection with sulphurous acid solution or dilute solution of iodine would be appropriate treatment.

TRACHEITIS.

Primary inflammation of the trachea is rare, the disease when found being usually an extension from the throat or bronchia, in which the morbid process attains a greater intensity than in the comparatively straight, roomy tube of the trachea, and these usually give their names to the affection—laryngitis, bronchitis.

Yet there are cases in which tracheitis will reach a high type, and in all cases of extension of inflammation from above or below the secondary disease—tracheitis—partakes of the character of the primary one. Thus the tracheitis may be simple inflammation from mechanical, thermal or chemical injury ; or it may be microbial, the weakened mucosa being attacked by saprophytic bacteria ; or it may be croupous ; or it may be diphtheritic ; or parasitic (from *sclerostoma*, *strongylus*, *filaria*, *linguatula*, *acarus*, *actinomyces* or *aspergillus*) ; or it may be from the growth of a neoplasm, benign or malignant ; or it may be specific (glanders, sheep-pox, tuberculosis, strangles, contagious pneumonia or influenza).

Lesions. In the simple inflammation the ramified redness betrays the general congestion, but there may be petechiæ, a swollen, velvety, friable condition of the mucosa, visible enlargement of the mucous follicles, more or less desquamation of the epithelium (the surface, columnar, ciliated layer, the middle pear-shaped or even the deep spherical layer). When the whole thickness of the epithelium has been removed the basement membrane shows clear, smooth and even glistening, while if the destruction has extended still deeper, distinct ulcers are formed.

Symptoms are not prominent yet the windpipe is tender to pressure at the affected parts, and in cases due to fracture of the rings, puncture, tracheotomy or the presence of a foreign body, this may be narrowly circumscribed. At first, with dry, thickened walls, there is a loud blowing sound in breathing, followed, on the advent of secretion, by a coarse mucous râle or gurgling. In very severe cases the exudate may infiltrate the muscular and subcutaneous connective tissue, causing, in solipeds, a distinct swelling along the line of the trachea, and inducing more or less difficulty in swallowing. Fever will correspond to the severity of the inflammation, there may be accelerated pulse and breathing and elevated temperature, or there may be little or no constitutional disturbance.

In the croupous or diphtheritic form the fever runs higher, dyspnoea is more marked, the breathing more stertorous, and later the discharge of shreds of false membrane in the expectoration is pathognomonic. In parasitic cases, the parasite may be found in the sputa, in malignant growths the microscopy of the expectoration may reveal the nature of the trouble, and in tuberculosis or glanders the testing with tuberculin or mallein will decide the case.

The *treatment* of these cases is essentially that of the laryngitis or bronchitis of which they are the concomitant. Localized cases may sometimes be effectively reached and treated through the opening made in tracheotomy.

DEFORMITIES OF THE TRACHEA IN SOLIPEDS.

Beside congenital distortions like truncated or double trachea, these are examples in acquired deformity in otherwise well developed windpipes.

Compression by an enlarged thyroid is a common and fatal condition in newborn lambs, and in one newborn camel I found the upper wings completely flattened upon themselves from side to side, so that it was impossible that breathing should be established (Goitre, Vol. III).

Detachment and wide separation of the free ends of the tracheal rings, I found in an old horse used for dissecting purposes. The space between the detached ends was sufficiently capacious to hold the œsophagus, with its attached nerves, vessels and lymphatics. Goubaud quotes a similar case. Cadeac one in which the ends of the ring were nearly four inches apart, and Vegezzi one in which the intervening space lodged the gullet, vagus and sympathetic nerves, and the carotid artery. In such cases it appears as if the tracheal ligament between the free ends of the rings were torn and the trachealis muscle detached from the outer free end for a considerable distance. The lumen of the trachea is invariably narrowed in its supero-inferior diameter, and the volume of air admitted must have been materially reduced.

Flattening of the trachea supero-inferiorly is usually the result of external pressure, and all flattening, causing as it does a deviation from the strictly circular outline, diminishes the calibre of the tube and its conducting power. Old horses with ewe-necks are especially subject to this distortion, from pressure of the vertebræ and their investing muscles. A sacculated œsophagus may be another cause, also tumors or phlegmons situated above the trachea (adenoma, melanoma, actinomycosis, strangles). Cadeac records a case of lateral flattening of the lower end of the trachea.

Excessive overlapping of the free ends of the rings is noted by Blaise, the rings appearing rolled on themselves around a narrow opening.

Thickening of the submucosa with constriction of the lumen is described by Lepper.

Elongated and tortuous trachea with successive deviations to the right and left are vouched for by Baldoni and Johné.

DISTORTIONS OF TRACHEA IN DOGS.

Much less common than in the horse, these are mostly due to goitres or to enchondroma, tubercle or epithelioma of the thyroid (Cadeac) or to other neoplasms along the course of the windpipe.

FRACTURES AND RUPTURES OF THE TRACHEA.

Fractures are rather common in *horses* from striking the trachea forcibly on the pole, shaft or in two-wheeled carts on the tailboard of the next cart in front. They are especially common in Scotland from the last cause, one man, driving two horses and carts, the second closely following the first, suddenly stops the first going down hill, and the second horse is driven by the impetus of its vehicle against the backboard of the first. Similar injuries have come from falling on the margin of the manger (Blaise), from bites (Bournay), kicks (Liquet), and from pulling on a halter tied around the neck (Reynal).

The common set of fracture is in the center of the ring, in the median line in front, and as a fibrous joint forms at this point, the circular outline of the ring is broken, the two detached halves of the broken ring come together at an acute angle, and by the contraction of the trachealis muscle above, the two sides are drawn together and form a long narrow ellipse with its long diameter supero-inferiorly. The resulting narrowing of the tube and the obstruction to the current of air is often extreme.

Laceration of the trachea by a staff, unskillfully used to relieve choking in an *ox*, is recorded by Ugheli, and laceration of the windpipe of a *goat* by the teeth of a shepherd dog by Riquet.

Symptoms. Immediately after the injury there is more or less effusion of blood which appears as a local swelling and perhaps as a sanguineous discharge from the nose. Emphysema of the connective tissue around the wound is often present and may extend to the surrounding tissues so as to cause extreme dyspnoea, and to crepitate on manipulation. This may be followed by abscess or local infection. The flattening of the broken ring from side to side and the prominent sharp angle of the line of union in front are diagnostic. In early and late stages alike the current of air is interrupted and a blowing or stertorous sound (roaring) is heard in accelerated or forced respiration. The excess of blood and exudate pressing on the vagus or recurrent nerve may cause extreme paralysis of the larynx with loud *roaring* or dyspnoea, and this may continue by reason of the organization and contraction of the exudate.

Treatment in the early stages may consist in incisions to procure the escape of the blood, gas and even of the exudate. Later the main resort is the performance of tracheotomy and the wearing of a tracheotomy tube.

ABSCESS OF THE TRACHEA.

This may occur as a localization of strangles, or as an extension of suppuration occurring in other tissues adjacent to the trachea. With its habit of causing absorption, and of advancing along the line of least resistance, the pus makes its way toward the free mucous surface of the windpipe, and bursting, discharges into its lumen, (Lafosse). In other cases it will dissect upward or downward between the chondro-fibrous outer coat, and the mucosa, and thus give rise to extensive, destructive and dangerous lesions. (Vogt, Raissac). The pus may in this way burrow as far as the mediastinum and even the lung, or again that which has escaped into the lumen may descend by gravitation into the bronchia, causing bronchitis or pulmonary abscess (Cadeac).

Symptoms. These consist in wheezing or stertorous breathing (roaring), and the presence of a hot, painful, perhaps fluctuating swelling around the trachea or to one side of it. In case an opening has been established into the trachea, compression of the swelling may empty its purulent contents into the lumen to the temporary relief of the dyspnoea and stertor. As the abscess is refilled these symptoms reappear. A resort to tracheotomy may give the means of a thorough exploration and a conclusive diagnosis. It may also be made subservient to a complete evacuation, disinfection and successful treatment of the abscess.

Treatment. Early lancing of the abscess, before a communication has been established with the interior of the trachea, is the most important resort. The sac may be treated with antiseptic injections daily until it has contracted and definitely healed. In case of extensive dissecting abscess, several openings may be necessary, and even the insertion of setons to secure a free dependent discharge and satisfactory healing. Rupture of the abscess into the trachea, or an extension by dissecting as far as the root of the lung is always to be looked on as a very grave

complication, because of the danger of resulting pulmonary abscess.

This is especially true of infecting abscesses like those of strangles. When the abscess has discharged into the trachea daily washing out of the bronchia with a dilute (2 to 3 vol.) solution of per-oxide of hydrogen will be indicated.

TUMORS OF THE TRACHEA AND BRONCHI.

Neoplasms are less common in the trachea than in the pharynx or bronchia. Fleming (1859) records a case of two polypi attached to the floor of the trachea in its lower third in a *horse* and almost completely closing the lumen, with thickening and osseous degeneration of the adjacent tracheal rings. The part was very tender, and exercise produced violent dyspnœa with a loud blowing sound. After months of suffering the animal died in a violent paroxysm following a drink of water. The arytenoid muscles were atrophied, the trachea filled with blood, and blocked by the polypi.

Parker records a case in a *mare*, in which an elongated, ovoid tumor, three inches long by over an inch in its short diameter, hung by a loose pedicle from the lower wall of the right bronchus, an inch below the trachea. Habitually this had blocked the right bronchus only, but when forced up by violent coughing, it lodged across the trachea blocking both bronchi and threatening suffocation. She died during the night from one of these paroxysms.

Jobert notes the case of a *bull* with a polypus as large as a hen's egg, attached by a thick, rigid, short pedicle to the floor of the trachea four inches in front of the lower end of that tube. He had violent paroxysms of coughing and dyspnœa, occurring at irregular intervals, and was butchered.

Watson records the case of a *horse* with a tumor extending through the tracheal walls, and bulging externally and internally, over an area more than five inches in length.

Siedamgrotzky quotes a case in the *horse*, of a colloid cyst growing from the mucosa immediately beneath the cricoid cartilage.

Gurlt and Gerlach respectively quote cases of polypi of the tracheal mucosa in cattle.

Cadiot describes two cases of polypi of the tracheal mucosa in *dogs*. They had hurried, difficult and anxious, wheezing breathing, lay on the sternum with head fully extended, and were attacked at intervals with paroxysms of violent coughing, dyspnoea, stertor, and vomiting. The tracheal mucosa was covered with red vegetations, over an inch in diameter, and composed largely of embryonal cells in a very vascular fibrous framework.

Nocard notes similar cases in the dog, the vegetations situated at the level of the bifurcation of the trachea and in one case extended well into the bronchia. Some of these cases lived for a year after the onset, and did not die of the disease but were killed by the impatient owner.

Merkle describes melanotic tumors of the trachea in a *horse*, and Hink, sarcoma—encephaloid of the windpipe of an *ox*.

Symptoms. The phenomena are somewhat alike in all these cases, the obstructed breathing, dyspnoea, cough, sometimes bleeding, extension of the head, and in case of loosely pediculated tumors intermittent paroxysms threatening asphyxia which may finally carry off the animal.

Treatment. The only resort in such cases is tracheotomy and extirpation in suitable cases, especially in pediculated tumors in the cervical region of the trachea. Sessile tumors, with a broad base implicating the whole thickness of the tracheal walls, may in certain cases prove inoperable unless a tracheotomy tube can be permanently worn below the level of the operation. Malignant growths are hopeless as a rule.

DISEASES OF THE CHEST.

Cough, its artificial production, precautions, character in different animals. Cough in disease, strong, full, ringing, weak, short, broken, abortive, dry, rasping, croupy, small, husky, soft, humid, rattling, mucous, paroxysmal, sympathetic, wheezing, roaring, whistling, grunt, moan. Expectoration, nasal in horse, also buccal in other animals. Morbid expectoration, watery, viscid, cloudly, flocculent, purulent, rusty, cretaceous, parasitic, foetid, varicolored, microbic. Expired air, warm, cool, vegetable odor, acid, foetid, heavy. Respiration, number in health, alteration in disease, rapid, slow, tardy, short, catching, quick, deep, labored. Position, standing, lying. Pleuritic breathing, broken-winded.

Before describing specific diseases, it is needful to consider the methods of physical diagnosis which enable the practitioner to differentiate the diseases of the chest. Some of the following remarks will bear equally on diseases of the nose and throat as well.

COUGH.

The cough so varied in health and in disease deserves careful practical study. It can usually be excited in solipeds, sheep and dogs by pinching the first ring of the windpipe between the thumb and first two fingers. In oxen it is best produced by compressing the anterior part of the larynx. In old cattle it is difficult to produce coughing. In no other animal should the attempt be made rudely nor unnecessarily repeated, as it may tend to excite or to aggravate already existing sore throat.

The cough of the healthy *horse* is *loud, sharp* and *ringing*, often repeated two or three times and followed by a snort (clearing of himself). It is weaker in young horses and shorter and drier in the aged.

The usual cough of the *ox* is *weak, dry, slightly husky* and *prolonged*.

That of the *sheep*, *small, weak* and *dry*.

That of the *dog*, also *weak* and *dry*.

A **strong, full, deep, ringing cough** is rarely heard in disease except in slight irritation of the larynx. In such cases the larynx is tender and light handling or pinching develops the cough.

A **weak cough** wanting in reasonance and heard only at a short distance from the horse, is usually associated with chronic

chest diseases and the last stages of acute thoracic inflammations.

A **short, broken or abortive cough** is one which appears to be suddenly cut short and suppressed, from the pain it causes. It is seen in the early stages of inflammations of the serous membranes of the chest or abdomen, when the quick rubbing of the dry and inflamed surfaces of these membranes on each other produces exquisite pain. It characterizes especially the *debut* of pleurisy, pleuro-pneumonia and peritonitis. This cough is infrequent for the same reason that it is short.

A **dry, loud, rasping, or croupous cough** is peculiar to the early stages of laryngitis, tracheitis and bronchitis, when the membrane is swollen, tense and dry. It is equally met with in diphtheritic and croupous affections implicating the larynx.

A **small, weak, dry, husky cough** without any *rasping* is characteristic of broken wind (heaves), emphysema of the lungs, asthma, or chronic bronchitis.

A **soft, humid or rattling cough** exists in the advanced stages of laryngitis, bronchitis and pneumonia when the activity of the inflammation has given way and a free exudation has taken place from the mucous membrane. It is usually accompanied by a discharge, in solipeds from the nose, and in other animals from nose and mouth.

A **soft cough** with a peculiar gurgling in the larynx is sometimes met with in croup.

A **paroxysmal cough** is one repeated five, ten, or twenty times in rapid succession. It is common in chronic bronchitis, early heaves, emphysema, verminous bronchitis and influenza. In such cases it is observed chiefly when the subject is brought out to the cold air, when he takes a drink of cold water, or when he has just had some active exertion, or some dusty or fibrous food.

A **symptomatic cough** is one due to disease in some other organs than the respiratory, and which irritates the air passages through nervous sympathy (reflex action). It is commonly small, short and dry. Inflammation or other disease of the liver, indigestions and intestinal worms are occasional causes of symptomatic cough. In the case of worms it may be loud, clear and ringing.

OTHER MORBID SOUNDS.

Besides cough, may be noticed the *wheezing* breathing, characteristic of *heaves*, *chronic bronchitis* and *asthma*, *roaring*, *whistling*, etc., as already described, and the sound between a *moan* and *grunt*, produced in pneumonia, especially in the ox.

EXPECTORATION.

This escapes almost exclusively by the nose in horses, because of the length of the soft palate. It may come from the mouth of other animals, especially when they cough. In the ox the discharge from the nose is rarely seen because of his licking it out with his tongue. Rattles (*râles*) in the larynx, trachea or bronchia, enable us to ascertain the source of such discharges.

The nasal discharge in acute catarrh, laryngitis or bronchitis, is thin, clear, and slightly viscid, becoming thick, whitish and flocculent as the disease advances. It is yellowish, thick, flocculent and intermixed with shreds of false membranes in diphtheria or in the croup of young foals and calves. It is clear, slightly viscid and watery at the onset of bronchitis. At the debut of pneumonia it is often reddish (rusty). It is bright, red, frothy and bloody in hæmoptysis. It is scanty, clear, watery, and containing minute white flocculi, in pulmonary emphysema (*heaves*.) It is white, thick, curdy, and devoid of viscosity in chronic bronchitis or when a pulmonary abscess is being emptied. It is grayish, thick and flocculent in advanced pneumonia in the horse.

Cows in the advanced stages of pulmonary tuberculosis expectorate a yellowish, sticky matter containing minute hard masses, often cretaceous. Calves and lambs suffering from *strongyli* in the lungs expel these in little pellets in the midst of a thick white material.

The expectoration is fetid, dark red and grumous in gangrene of the lungs.

In pulmonary tuberculosis and glanders the expectoration usually contains the respective bacilli.

CHARACTER OF THE EXPIRED AIR.

The breath is sensibly warm in excited breathing, high fever and acute bronchitis and pneumonia. It is cool in most chronic

diseases, in advanced consumption and hydrothorax. Its odor is vegetable and acid in the acute indigestions of cattle, and fetid in many chronic diseases of the air passages attended with destruction of tissue, or the escape of imprisoned pus, but especially fetid in gangrenous sore throat, or gangrene of the lung.

MODIFICATION OF THE RESPIRATION.

The number of respirations in a given time may afford valuable indications in the horse but in the other domestic animals variation in number imports little. In the ox for instance, the respirations in health may vary from twelve to eighty per minute, according to the heat of the cowhouse, the plentitude of the abdominal organs and other circumstances. So in the sheep and dog slight causes, quite compatible with health may cause the breathing to become short, panting and hurried.

The young horse breathes ten to twelve times per minute, the adult animal nine to ten. Any excitement accelerates. A horse walked a few hundred yards had the respirations increased from ten to twenty-eight per minute ; after trotting five minutes they numbered fifty-two ; after galloping five minutes sixty-five.

Hurried breathing occurring independently of exercise, heat of the atmosphere, or distension of the abdomen, is indicative of fever, especially if associated with rapid pulse and increased heat of the body.

Infrequent respiration appears in certain brain diseases in the intervals between the more violent paroxysms, also in poisoning by opium and other narcotics. **Tardy or slow respirations** differ from those last noticed in the act occupying a longer time. In infrequent breathing the act may be short, though there are few respirations in the minute. This is likewise seen in brain diseases and sometimes in heaves. In the last case there is **double action of the flank**, each act of expiration being effected by two successive and distinct elevations of the flank.

Quick breathing in which the act occupies only a short time is usually abruptly cut off, the inspiration terminating by a catch or jerk. It is significant of the early stage of pleurisy, and arises from the desire to avoid the pain attendant on the rubbing together of the inflamed surfaces during deep inspiration. It is further seen in tetanus, peritonitis and pleurodynia.

Deep breathing with great lifting of the flanks and loins is characteristic of water in the chest, and consequent inability to inflate the lungs.

Labored breathing, which is at once hurried, deep, and without intermission, is seen in severe laryngitis, croup, capillary bronchitis, and pneumonia, in all cases alike from the difficulty experienced in introducing into the lungs the requisite amount of air. It is especially marked in double pneumonia, pleuropneumonia, complicated with effusion in the chest, and in old standing heaves with dilatation of the right heart.

In all such cases where there is much interference with the æration of blood, whether from obstruction to the circulation of blood or a hindrance to the introduction of air, the horse invariably stands. The fact that he has lain down may be taken as an indication that improvement has taken place. The peculiarity is due to the sharp outline of the horse's sternum inferiorly so that in lying down he is compelled to rest on his side and the whole weight of the body tends to compress the chest. In the ox, sheep, pig and dog, which can rest on the sternum, breathing can be carried on with comparative ease in the recumbent position, and these animals accordingly do not necessarily stand except in very extensive and violent affections of the chest.

The occurrence of a short inspiration suddenly checked and a prolonged expiration characterizes pleurisy, the check to the inspiratory act being because of the pain caused by dilating the thorax.

The double lifting of the flank in expiration :—the act appearing to be performed by two distinct and successive acts is one of the most prominent symptoms of heaves, but it is not peculiar to this disorder. In the horse it exists in chronic bronchitis, dilatation of the right heart, old standing hydrothorax, and diaphragmatic hernia. It is further frequent in the acute diseases of the chest. In oxen it accompanies pulmonary emphysema, pulmonary consumption, dilation of the heart, foreign bodies in the heart, and dropsy of the pericardium.

If accompanied by clear resonance over the chest, a permanent wheezing noise heard over the ribs, and the small, weak wheezy cough, it indicates emphysema (heaves, broken wind). If with strong impulse of the heart against the ribs behind the elbows,

venous pulse in the jugulars, and modification of the second sound of the heart, it bespeaks cardiac dilatation or other heart disease. If with paroxysmal cough, white, flocculent, nasal discharge and harsh rasping sounds heard at the lower part of the trachea or along the upper part of the lungs it betrays chronic bronchitis.

RELATIVE POSITION OF THE LUNGS, HEART AND OTHER ORGANS IN THE DIFFERENT DOMESTIC ANIMALS.

Relative positions of thoracic organs. Diaphragm, heart, lung, in horse, ox, sheep, pig, dog. Palpation.

The *chest* is that portion of the trunk closed in on each side by the ribs, above by the bones of the back, below by the breast bone, and behind by the diaphragm. It forms thus a cone flattened from side to side anteriorly, and with its base, represented by the diaphragm which slopes obliquely from above downward and forward and bulges forward in the center to a greater or less extent according to the plentitude of the stomach and bowels. It results from this arrangement of the diaphragm that a very thin layer of lung only reaches to the posterior part of the chest, and that beneath this are solid and hollow abdominal organs which modify the results of physical examination.

In the **Horse** the anterior third of the chest is covered laterally by the bulky and muscular shoulders so that it cannot be satisfactorily examined. In the median line of the chest, at a point corresponding to the third, fourth, fifth and sixth intercostal spaces, is lodged the heart. It deviates slightly to the left side below and by virtue of a notch in the lower border of the lung is enabled here to reach the surface and its beats may be felt by the hand laid on the side of the chest just behind the left elbow.

In the **Horse** the diaphragm is attached by its outer border to the last rib, and to the lower ends of all the asternal ribs, and the extremity of the breast bone. A thin layer of lung accordingly extends to between the two last ribs superiorly and down to near the lower end of the asternal ribs. The subjacent abdominal organs are arranged as follows :—*On the left side*, and count-

ing from below, the large intestines (double colon), the stomach and spleen and a portion of the left lobe of the liver :—*on the right side*, below, the large intestines, above, the liver and pancreas. Of these the stomach and intestines frequently contain gases, while the liver by its solidity gives a special solid character to the right posterior part of the chest. The spleen is too deeply situated to effect much the results of a physical examination. The greatest substance of lung is between the upper and middle thirds of the thorax. The anterior third is inaccessible on account of the shoulders, but more than usual may be reached by raising the fore limb and drawing it forcibly forwards. The space between the third and seventh ribs is occupied by the solid mass of the heart, which especially modifies the result of physical examination on the left side where a notch in the lung allows it to approach the surface.

In the **ox** the diaphragm is only attached to the last rib for two or three inches at its upper extremity ; it is fixed to the second last rib as far down as about one-third of its length ; thereafter it is attached in succession to the middle third of the third last, to the lower third of the fourth last, to the lower ends of the next two in succession and to the sternum. The result is that the lungs do not extend so far back relatively to the ribs as they do in the horse. They are virtually absent from the last intercostal space, present only in the upper third of the second last, in the upper two thirds of the third last and reach the lower third only in the space between the ninth and tenth ribs. The paunch alone occupies the space beneath the asternal ribs on the left side, and the liver and the solid mass of the omasum and the abomasum that beneath the right. The shoulders in fat improved beef breeds absolutely prevent examination of the anterior third of the chest, though in thin animals and dairy breeds and scrubs more of this may be exposed by raising the fore limb than in the horse. The heart corresponding in position to the third, fourth and fifth intercostal spaces is more completely covered by lung tissue and does not strike the left side so forcibly as in the horse.

In the **sheep** the lung extends to the last intercostal space, nearly as far as its lower end and the heart is covered on the left side as well as on the right by lung tissue. The shoulder is very movable and unless in very fat animals allows of an examination of the greater part of the anterior third of the chest.

In the pig, fat and indocility combine to defeat our purpose in examination of the chest. If these can be obviated it is well to know that the diaphragm is attached to the upper two-thirds of the last rib, and to the next three in front above their lower third.

In the dog, the diaphragm is attached to the upper two-thirds of the last rib, to the lower third of the next and to the lower ends of the two following and to the breast bone. The shoulders are so mobile and the breast bone so thin that nearly all the chest may be satisfactorily examined. The heart, covered on both sides by lung, lies nearly horizontally on the breast bone, through which its position and bulk may be clearly made out by percussion.

EXAMINATION BY TOUCH.

Pressure by the fingers in the spaces between the ribs corresponding to the pleura will cause flinching and perhaps grunting in pleurisy. The same result will be seen in pleurodynia. In hepatized lung and pleurisy with adhesions there is a diminished sense of the movement felt in the intercostal spaces of the part in health.

PERCUSSION.

Methods. Tissues as good and bad conductors of sound. Immediate, mediate percussion. Bilateral symmetry and divergence. Effect of building, race, etc. Horse, left side, right. Ox, left side, right. Effect of 1st and 3d stomachs, liver, etc. Sheep, diaphragm, heart. Pig, fat, lean, heart. Dog, method. Birds, back, ribs. In disease, increase, decrease, absence of resonance, in large area, in patches. Crack pot sound.

This consists in striking the walls of the chest so as to bring out the resonance of the parts. In proportion as we tap gently with the tip of the finger or strike forcibly with the closed fist will we elicit the sounds from the superficial or the deeper parts of the lung. Hence slight blows only must be used where the lung tissue is thin, to avoid bringing out the resonance from the deeper seated organs, and both must be resorted to where the lung is thick to ascertain its condition at the various depths. Where a moderate force is requisite the four fingers and thumb of the right hand are brought together in a line and the weight of the hand as moved from the wrist is employed to bring out the sound. The ribs being hard convey sound best from the deeper parts, and on them percussion is usually made. Care should be taken not to mistake the lesser resonance conveyed through the soft tissues of the intercostal spaces for an indication of a diseased condition. In proportion too as the ribs are covered with flesh or fat, the resonance will be diminished and a stronger blow will be necessary to bring out the sound from the lungs.

If a blow is made directly on the side of the chest the percussion is called *immediate*; if made upon an elastic solid body (pleximeter) laid on the outside of the chest it is *mediate*. The readiest and perhaps the best pleximeter is the middle finger of the left hand which is to be applied flat upon the side of the chest to receive the blow directed perpendicularly to its surface. In fat or fleshy subjects it should be pressed firmly on the surface so as to compress and condense the soft parts and render them better conductors of sound. Some use flat pieces of ivory, silver, caoutchouc but in employing these the nails of the right hand must be carefully pared, lest by striking the solid body they produce a sound which interferes with the true pulmonary resonance.

In examining the chest the two sides should be compared and if allowance is made for the dullness felt in the lower half immediately behind the left elbow caused by the position of the heart, and the deadness of the sound on the last few ribs on the right side where the liver is situated, further deviation from a bilateral symmetry of sound is indicative of disease. The general resonance will be decreased by a full stomach which prevents the full inflation of the lungs, and it will be increased if the animal stands on a wooden floor with an empty space below. A short statement of the degrees of resonance over the different parts of the chest in the various races of the domestic animals in a state of health may prove useful.

Horse.—Left side. In the upper third the resonance is full behind the shoulder. It diminishes from the 13th rib backward and from the decreasing thickness of the lung the blows should become less and less powerful. In this space forcible striking brings out the drum-like resonance of the abdominal organs.

In the middle third the sound over the 5th and 6th ribs is distinct but not full; it increases to the 11th rib and then decreases to the last.

In the lower third a very slight resonance may be observed over the 4th rib; over the 5th, 6th, and 7th, where the heart approaches the surface the sound is dead; while from this to the 13th rib a slight resonance may be made out.

Right side. The upper third resembles that on left side from the shoulder as far back as the 13th rib, behind which anything above the gentlest blows brings out a drum-like sound from the large intestine (double colon) especially. This is clear when that is distended with gas.

In the median third the resonance resembles that on the left side. In the lower third it equally corresponds as far as the seventh rib, behind which the sound is dull because of the proximity of the liver.

Ox.—Left side. The upper third is clear in sound from the eighth to the tenth ribs, and behind this by gentle tapping to the second last (twelfth). Forcible striking, however, brings out the drum-like sound of the upper sac of the paunch which always contains more or less gas.

The middle third has a clear resonance as far as the seventh

rib ; this diminishes to the ninth, behind which it is usually replaced by a dullness due to the presence of food in the anterior part of the paunch. By drawing back the limb percussion may be employed over the first and second ribs as well.

In the lower third the first two ribs can be examined and a clear sound should be educed. On the 4th, 5th, and 6th ribs there is a full resonance, the heart being here covered by lung tissue, contrary to the condition in the horse. From the seventh the sound becomes duller and the dead sound from the food in the rumen characterizes the lower fourth of the ninth rib.

Right side. From the shoulder the resonance gradually decreases in the upper third to the eleventh rib, beyond which the sounds obtained are only from abdominal organs. In the middle third considerable resonance is met with over the first and second ribs, it is very full and clear over the 5th, 6th, and 7th, whence it decreases and is quite lost behind the 10th. In the lower third a clear sound can be elicited over the 1st, 2d, 4th, 5th, and 6th ribs ; this is lessened over the 7th and 8th, and completely lost behind the 9th. Any but the slightest blows over these three last ribs brings out the dull, solid sound from the liver.

A very full paunch greatly increases the anterior convexity of the diaphragm, and compresses the lungs into the anterior part of the chest. If the contents of the rumen are solid the resulting dullness on percussion might be mistakenly supposed to indicate consolidation of the lung. This source of error must be carefully guarded against.

Sheep. Percussion in the sheep differs from that in the ox chiefly in the following particulars : The diaphragm being attached to the last rib, as in the horse, the diminishing resonance of the lung may be traced as far back as in that animal. Thus a pulmonary sound can be obtained in the upper third as far as the last intercostal space, in the middle as far as the second last, and in the lower as far as the fourth from the last. Over the lower part of the fifth and sixth ribs on the left side the resonance is remarkably clear owing to the great relative thickness of the anterior lobe of the left lung which here covers the heart.

Pigs. In fat pigs the results are almost negative. In lean animals the middle third on each side gives out a clear resonance behind the shoulder as far as the seventh rib, from which it diminishes to the second last (thirteenth). The sound is less

clear in the upper and lower thirds. On the fifth intercostal space below, and on the left side the sound is dull owing to the exposure of the heart through a slight notch in the lung.

Dog. Percussion is very satisfactory in this animal because of the amplitude of the chest, the thinness of its walls and the small bulk of the abdominal organs. In the upper and middle thirds on both sides alike the sound is clear and full as far back as the seventh rib, whence it decreases to the last. In the lower third a distinct but moderate sound marks the first eight ribs and is equally clear on the right and left sides. The thinness of the lung in its posterior part demands that percussion be effected by the middle finger only, without any movement of the hand. Unless the dog is very fat, good results may be obtained by percussion over the first and second ribs, the shoulder blade and breastbone.

Birds. In these and especially in the webfooted (ducks, geese,) the sternum is so thickly covered by flesh that no result can there be obtained. Beneath the wings, however, and upon the back, percussion through the medium of a small coin as a pleximeter and with the middle finger alone, is valuable. Beneath the wing a clear sound may be drawn out over nearly all the ribs and on the back over a less extent (two and a half to four inches, according to size).

PERCUSSION IN DISEASE.

Increase of resonance without any perceptible modification in character is usually partial and depends on the increased distension of the air cells of one lung, or part of a lung, to make up for the loss of a part or a whole lung through hepatization, or pressure by false membrane or from water in the chest. If a part of a lung is solid and impervious it gives a dull, dead sound, contrasting strongly with the increased clearness of the remainder. So with water in the chest, the clearness of the upper parts contrasts unmistakably with the dullness of the lower. By watching the advance or retirement of these symptoms the solidification of a lung and its process of clearing up, and the effusion of water in the chest and its removal may be equally traced through all these stages.

If the increased clearness is confined to the upper, lower, or

posterior border of one or both lungs, the sound being natural over all other parts, it indicates the existence of emphysema of the lungs, a condition almost constant in horses with heaves.

If the sound is drumlike over most of the lung it is due either to extensive emphysema or to the presence of air as well as liquid in the cavity of the chest. In the case first noticed there will be the double action of the flank, the weak, dry, husky cough and the wheezing breathing; in the last there will have been the previous attack of pleurisy, and the application of the ear to the chest will detect a splashing sound constant or heard only at intervals or on rising. This should be carefully distinguished from abdominal gurgling.

Diminished resonance, noticed over an entire lung, may be due to congestion or œdema of the lung, to the formation of a thick false membrane over the inner surface of the ribs or to a false membrane enveloping the lung and preventing its due distension. Congestion will be distinguished by the blueness of the mucous membranes and the presence of a crepitant sound heard on auscultation. Pleurisy is known by the tenderness on percussion or on pinching the intercostal spaces, and by the presence in many cases of a friction sound. The sound may be further lessened in cattle by the deposit of tubercle on the inner side of the ribs, or the extensive deposition of miliary tubercle throughout the substance of the lung.

Absence of resonance, the sound brought out by percussion being similar to that obtained by practising it over the muscular masses of the haunch, is always partial. It is due usually either to hepatization or to water in the chest. Hepatization is distinguished by its rarely affecting the lower thirds of both lungs at once, by the presence of a crepitating râle round the margin of the area of dullness, and by the increased resonance and respiratory murmur over the sound parts of the same and the opposite lung. In water in the chest, on the other hand, a friction sound and much tenderness precedes the dullness; the tenderness continues and the dullness reaches the same height on both sides of the chest, in the case of the horse. In the ox, water may exist on one side of the chest only, but the tenderness on pressure and the absence of any crepitation serve to distinguish the case from pneumonia. In the smaller animals the position of the dullness may be altered by

turning the patient on its back as the water always gravitates to the lowest point.

The presence of extensive deposits of tubercle, of cretaceous material in tubercular cows and sheep, and the presence of large cysts in the lung may give rise to dullness over a circumscribed area. Such areas of dullness are usually multiple with sound lung between.

A further modification on percussion is known as the **cracked pot sound** is sometimes heard in horses and cattle. It may be aptly represented by laying the palms of the two hands together in such a way that they meet all round and leave an interval filled with air right in the centre. The back of the one hand is then struck against the knee when the noise of the air escaping gives the characteristic sound. It occurs in consumption or in the advanced stages of inflamed lungs when a large tubercle or abscess has burst into a bronchial tube and the resulting cavity opens into this tube by a narrow orifice.

AUSCULTATION.

Mediate and immediate auscultation. Methods, quiet, normal chest sounds, tubal, bronchial, vesicular, respiratory, cardiac. Juvenile respiratory murmur. Horse, left side, right. Ox, left side, right. Accidental sounds, rumbling, gurgling, crepitation, friction. Sheep, special features. Goat, force. Pig, Dog, Birds, morbid chest sounds. Increase, general, partial. Decrease, general, partial. Absence. Bronchial sound in excess, in improper place. Cavernous, amphoric, mucous sounds. Râles, sonorous, sibilant, mucous, submucous, crepitant, subcrepitant. Creaking, metallic, tinkling, gurgling, splashing, friction. Timbre of Cough. Palpitation. Mensuration.

This is a term used in medicine to denote the mode of exploring an organ by applying the ear over the region in which it is situated and deducing the healthy or diseased condition by the sounds heard. First employed by Lænnec in human medicine it was quickly availed of for the lower animals by Delafond and Leblanc.

Auscultation is **mediate** or **immediate**. **Immediate Auscultation** is practiced by applying the ear directly upon the skin, either bare or covered with a handkerchief. In **Mediate Auscultation** an instrument called a stethoscope is employed to convey the sound from the surface of the body to the ear of the examiner. The common stethoscope is formed of soft wood (cedar or ebony) or of gutta percha, is from five to seven inches long and a quarter of an inch in the bore. The end applied on the skin is widened into a funnel three-fourths of an inch across at the mouth ; the opposite end is flattened out to apply to the ear, is about two inches in diameter and has a hole in the centre to convey the sound. A flexible stethoscope is also used with either one or two ear pieces and though less convenient in general than the common variety possesses this advantage when the heart is being examined ; that it conveys the sound without the impulse of that organ.

In immediate auscultation the ear should be closely applied to the surface, the right ear being used for the left side and the left ear for the right, but a preference should always be exercised in favor of that in which the sense of hearing is most acute. If a handkerchief is used, a single fold only must be applied, otherwise the two layers may rub on each other and produce distract-

ing sounds. In mediate auscultation the instrument should be held perpendicularly to the surface, accurately applied alike to the skin and the ear, and pressed firmly on the surface to condense the soft structures beneath the skin and render them more conducting. If held by the hand care must be taken to avoid the slightest movement of the fingers on the stethoscope, and long hairs should be prevented from entering the tube as being likely to produce additional sounds.

Among other points the following must be attended to in auscultation. Avoid a position in which the animal can strike you with its hind limbs. If necessary in irritable or ticklish subjects have one fore leg held up. Select a quiet time and place, early morning or night is usually best. Endeavor to protect the patient from the irritation of insects or the examinations may be fruitless. Never auscultate over a contracting muscle ; the sound of muscular contraction will prevent a correct result. If the natural sounds are indistinct increase them by exercise. The smaller animals are examined with the greatest facility standing upon a table or held in the upright posture with the body resting on the thighs or on the hind feet only. Birds can be held by the wings which may be raised and drawn inward towards the median line to expose the back and sides of the chest.

HEALTHY CHEST SOUNDS.

In all healthy animals two distinct sounds are heard over the chest:—the **tubal** or **bronchial** sound, and the **vesicular** or **respiratory murmur**. The **bronchial** sound caused by the air sucking through the larger bronchi is best heard by applying the ear to the breast over the lower end of the windpipe or to the upper third of the chest immediately behind the shoulder. The **respiratory murmur** is clear and full in the middle third of the chest immediately behind the shoulder. It is louder and more prolonged in inspiration than in expiration and in the right lung than the left, especially in cattle and sheep in which the former is more capacious. It is louder in young animals than in old, hence the name of **juvenile** respiration applied by Leblanc. In thin animals it is better heard than in fat ones, the chest walls being thinner, firmer, and more conducting. In animals of a

nervous temperament like the English racer it is more distinct than in the Percheron, Clydesdale and other heavier breeds. Deep, broad capacious chests emit a stronger sound than such as are shallow, narrow and short. Exercise, fear, or any excitement accelerating the respiratory act increases the sound. A full stomach, certain narcotics and other depressing influences lessen it. Other things being equal the sound is lower in cattle and sheep than in other domestic animals.

Horse. The ear pressed strongly upon the breast where the windpipe enters detects a strong blowing sound referable to the lower end of the trachea and the bronchi. In young foals a respiratory murmur is heard when the stethoscope is applied in front of the shoulder, the limb being meanwhile drawn backward.

A similar murmur may be heard, but less distinctly over the shoulder blade at this age.

Left side. *Behind the shoulder* in the *upper third* of the chest the sound is loud and somewhat harsh, the respiratory murmur being here supplemented by the noise of the air rushing through the larger bronchia. From the 13th rib the respiratory sound alone is heard and becomes weaker to the second last (17th).

In the *middle third* the respiratory murmur is moderately clear from the 4th to the sixth rib, it becomes louder and clearer to the 6th from which its force gradually diminishes and is lost over the 16th. In the *lower third* over the 4th, 5th, and 6th ribs the respiratory sound is replaced by the sounds of the heart, each beat being distinctly divided into two sounds, the first dull and prolonged, the second short and quick. The respiratory murmur is heard over the 7th and 8th ribs, is weaker on the 9th and lost over the 10th. In the middle and lower thirds, but especially towards the posterior part of the chest, abdominal sounds are often heard. They consist chiefly in gurgling or in a noise like that caused by the air rushing into a bottle which has been turned upon its side when full of water. Such sounds are easily distinguishable from those occurring in a diseased chest as they bear no relation to the rhythmical action of breathing.

Right side. In the *upper and middle thirds* the sounds do not differ from those of the left side. In the *lower thirds* the respiratory sound is clear from the 4th to the 7th ribs; from this it decreases and is lost at the 10th.

Ox. In very lean cattle the respiratory murmur heard in front of the shoulder and over the scapula is more distinct than in the same region of the horse.

Left side. In the *upper third* a clear respiratory murmur is heard over the 8th, 9th and 10th ribs but is lost about the 11th.

In the *middle third* the vesicular sound is feeble at the lower margin of the region and immediately behind the shoulder because of the proximity of the base of the heart. Towards the upper margin it is loud and harsh being complicated by the *tubal* sound. It is full and clear over the 7th rib whence it decreases in force to be lost at the 11th above and the 10th below. In the *lower third* the double heart beat alone is heard over the lower part of the 4th rib, the respiratory murmur reappears over the 5th and 6th whence it becomes weaker and is lost at the lower and upper margins of the region respectively over the 8th and 9th ribs.

Right side. The sounds of the *upper third* simply repeat those of the left side. In the *middle third* the chief difference is the greater clearness and strength of the respiratory and tubal sounds immediately behind the shoulder. In the *lower third* a moderately strong respiratory murmur is rendered harsh by a tubal sound due to the proximity of the large bronchus going to the anterior lobe of the right lung. The respiratory murmur continues with diminishing force to be lost over the 8th and 9th ribs.

Accidental but healthy sounds. These are more loud and frequent in the ox than in the horse. There is the same irregular rumbling and gurgling especially on the posterior parts of the chest. Gurgling as from a full bottle inverted is often clearly heard over the last six ribs on the left side, and appears due to the passage of liquids between the paunch and honey comb bag. An occasional sound as of water falling into an empty barrel is heard in the same region in cases of slight tympany and after saliva has been swallowed. Rumbling sounds are chiefly heard over the last ribs on the right side where the large and small intestines are situated. The superadded sounds in the ox are those of *crepitation* and *friction*. The *crepitation* or fine crackling due to a dryness of the areolar tissue under the skin is frequently present in oxen in average health. A fine *crepitation* is also heard on the left side from the bursting of myriads of minute bubbles

of air generated among the contents of the paunch during the process of digestion. This is especially marked after the animal has fed on green food or potatoes. A loud *friction* or *rubbing* sound, which may be imitated by placing the back of one hand upon the ear and rubbing the palm of the opposite hand upon it, is likewise heard over the left side after eating. It is produced by the movements of the paunch during contraction and not being synchronous with the respiratory acts cannot be confounded with the friction sounds of pleurisy.

Sheep. The diaphragm being attached to the last rib, as in the horse, the respiratory murmur may be heard to the second last. The shoulders being more movable than in the ox the anterior part of the chest can be more satisfactorily examined. The vesicular murmur is heard along the whole lower third on the left side though the heart sounds are superadded over the 4th, 5th and 6th ribs. Crepitation from the subcutaneous areolar tissue is rarely heard. Otherwise the sounds of the chest and abdomen correspond to those of the ox.

Goat. This animal differs from the sheep mainly in the greater force and clearness of the respiratory murmur.

Pig. It seems ridiculous to speak of auscultating the pig, yet he is sometimes thin enough and quiet enough to permit of one obtaining satisfactory results. Gentle treatment and scratching the back and abdomen will often persuade him to be temporarily quiet and docile. The vesicular murmur is very clear in the middle third of the chest on either side, but diminishes gradually on the last six ribs, and disappears on the second last. It is much less intense in the upper and lower thirds. In the posterior part of the chest rumbling and gurgling abdominal sounds are frequent.

Dog. The respiratory murmur is very clear over the whole chest. It is most intense along the middle third and becomes less clear on the four or five last intercostal spaces. The mobility of the shoulder permits an examination of nearly the entire chest. The respiratory murmur may be heard over the entire length of the lower third on the left side though the heart's sounds are equally heard over the 4th, 5th and 6th ribs. Rumbling and gurgling abdominal sounds are much less frequent than in herbivora and omnivora.

Birds. The respiratory murmur is loud, clear and almost harsh on the sides of the thorax, beneath the wings, and considerably softer as heard on the back.

MORBID CHEST SOUNDS.

The close study of the healthy chest sounds upon the living animal is an essential prerequisite to the appreciation of the morbid. The abnormal noises are so varied, merge into each other by such imperceptible degrees, and so coexist and complicate each other that they often prove extremely puzzling to the unpracticed ear. It is no more necessary that the musician should educate his ear to appreciate the most delicate gradations of musical notes, than that the auscultator should educate his in the sounds of the healthy and diseased chest. Written instructions are of about equal value in the two cases, they prove auxiliaries in the acquisition of knowledge but they can never supersede the practical study of the chest. A mere theoretical knowledge is too often useless in the presence of the patient.

The abnormal chest sounds are either modifications of those existing in health, or superadded sounds which have no counterpart in the healthy chest.

Modifications of healthy sounds. The **vesicular** or **respiratory murmur** may be **increased** or **diminished** in force or it may be entirely absent.

Increase of the respiratory murmur, is merely an increase in force without any modification in character and resembles *juvenile* respiration. If increased equally over the entire chest it is **general**, if only in part it is **partial**. **General increase of the vesicular murmur** is heard after an animal has been submitted to moderate exertion for ten or fifteen minutes. In animals at rest it is heard in active fevers and in the symptomatic fever which attends acute inflammation.

Partial increase, as for example in one lung only, or in circumscribed parts of both lungs, and especially along their superior borders, is indicative of disease of the lungs or the pleuræ. It testifies to the impermeability to air of some other portion of lung, from congestion, splenisation, hepatisation, plugging of a bronchial tube with tenacious mucous, tubercular deposits, tumors, emphysema, or hydrothorax. (See under these names.) The

healthy portion of lung in such cases takes on the function of the whole, and the loud breathing is called **Supplementary**.

Diminution of the respiratory murmur, like its increase, may be **partial** or **general**. **General diminution** is seen in anæmia, in low fevers, in all very prostrate conditions from the mere want of power to dilate the chest ; in general pulmonary emphysema (broken wind, heaves), in general miliary tubercular deposit in the lungs, or in that form in cattle in which the tubercle has been replaced by cretaceous deposits, from the animal's inability to fully dilate the air cells ; in enteritis, peritonitis and metritis the chest is less fully dilated because of the pain attendant on that act, and the breathing being short and quick the murmur is correspondingly low. In certain brain diseases with sluggish respiration the sound is equally feeble.

Partial diminution of murmur is more surely indicative of lung disease. It may arise from partial congestion when a supplementary murmur will be observable over other parts of the lungs, and a crepitant râle soon appears in the congested part ; from local emphysema in which there is increased resonance in percussing the part ; from tubercular or cretaceous deposit, when there will be exaggerated murmur elsewhere ; or from bronchitis with blocking up of one or more small bronchial tubes and with louder respiratory sound in other parts.

Absence of respiratory murmur may be due to various causes, all of a diseased nature. Hepatisation of lung may be recognized when this condition is found associated with a crepitating râle around the margin of the silent part, and when percussion shows its solidity and want of resonance. Splenisation is associated with absence of respiratory sound and dullness on percussion, but no surrounding crepitation. Absence of sound in water in the chest is confined to the lower part of the thorax, keeps the same level and ratio of increase in front and behind, and in the horse on the two sides, and has been preceded by the characteristic catching breathing and the friction sounds of pleurisy. Large tumors and extensive and circumscribed tubercular deposit will give rise to absence of sound over a limited area, and plugging up of one or more bronchial tubes will lead to a similar result. Hepatisation of lung, and water in the chest are, however, the common causes of loss of respiratory murmur.

The **bronchial or tubal sound** may be increased in pitch and in harshness in two conditions. 1st. In the early stages of bronchitis when the lining mucous membrane of the air passages is dry, thickened and inelastic. 2d. When that portion of lung intervening between one of the larger tubes and the surface of the chest is solid (hepatised) and thus proves a better conductor of sound than in the normal condition.

Superadded abnormal sounds. The **bronchial** sounds may be altered in their character so as to become **cavernous**, **amphoric** or **mucous** (rattling). The **cavernous** sound is usually caused by the presence in the lung of the cavity left after the discharge of an abscess or softened tubercle into a bronchial tube. It is thus preceded by cough and white, creamy discharge from the nose. If the discharge is fetid and grumous there has probably been circumscribed gangrene of the lung. An approximation to the sound may be produced by blowing into a wide-mouthed glass or porcelain vessel. The sound of **amphoric respiration** on the contrary is like that made by blowing into a narrow-necked bottle. It is due to a similar cavity with a small orifice or to the existence of pneumo-thorax communicating by a narrow canal with a bronchial tube. It is rare in the lower animals, but Delafond mentions one case in the horse and two in dogs.

Rales. The remaining morbid sounds are known as *râles*, or rattles. They may either be referable to the bronchial tubes or the lung tissue. They are called dry or humid, according as they convey the idea of air drawn through a dry tube or one containing liquid.

The **dry rales** are due to narrowing of the bronchial tubes from the pressure of adjacent tumors, the thickening of the mucous membrane or the deposition on the surface, of layers of tenacious mucus. The greater the narrowing the shriller the sound, and hence the distinction of *bronchial râles* into **sonorous** and **sibilant** (whistling).

The **sonorous rale** has been variously exemplified by the humming of a gnat, the cooing of a wood pigeon or the bass notes of a violin. It commonly bespeaks the onset of bronchitis and testifies to the thickened, dry and rigid character of the tubes, but may give place in as short a time as three hours to a mucous *râle*

from the occurrence of a free secretion. It rarely extends over two or three days. Sometimes when caused by a piece of tenacious mucus obstructing a tube, it is very transient, disappearing at once when the mucus is expelled by coughing. Sometimes it is modified by an occasional clicking sound from the flapping of a shred of semi-solid mucus attached to the walls of a bronchial tube. This disappears when breathing becomes more hurried.

The **sibilant** (whistling) **râle** often depends upon the same causes as the sonorous, but implies a narrower closure of the tubes. More frequently it is heard further back on the chest and results from pulmonary emphysema and dilation of the smaller bronchial tubes (broken wind, heaves). It is then heard chiefly in expiration and coincidently with the second quick lifting of the flank. It is further associated with the double lifting of the flank in expiration, with the short, weak, paroxysmal cough and the indigestion characteristic of heaves. If the whistling noise is so loud as to be heard without applying the ear to the chest it is called wheezing.

A **mucous râle** is caused by air passing through any liquid contained in the bronchial tubes, such as mucus, pus, or blood. It may be imitated by blowing a large number of soap bubbles in a thick lather and noticing them burst simultaneously or successively. It is chiefly observed in bronchitis after the preliminary dry stage of the mucous membrane has passed off and an abundance of mucus has been secreted. The nature of the sound will vary according as it comes from the larger or the smaller tubes or in other words as the bubbles are large or small. That from the smaller tubes is sometimes called a **sub-mucous râle**. Either of these râles may be temporary or permanent as the mucus may be momentarily cleared away by coughing.

The **crepitant râle** is a sound of very fine crackling which has been variously compared to the crackling of salt when put on red hot coals, the noise of a sponge expanding in water and the rubbing of a small lock of hair between the finger and thumb close to the ear. The existence of the *crepitant râle* usually denotes the existence of the early stage of inflammation of the lungs, and the progress of hepatization in such cases may be traced by the advance of the line of crepitation which precedes it. So the pro-

gressive absorption of exuded matter in recovery may be equally followed by a circular line of crepitation gradually decreasing in area until it meets in a point. The observations will be corroborated by the dull sound elicited on percussing the parts. The production of the sound has been attributed to the passage of air through the thick mucus in the smallest bronchial tubes or more plausibly to the separation of the walls of the air sacs and cells during inspiration, they having been previously adherent by reason of the secretions.

Crepitation is not heard in all pulmonary inflammations. In weak animals with a low type of inflammation tending to gangrene, and in those cases of broncho-pneumonia in which a viscid mucus blocks up the bronchial tubes passing to the affected lobes, it may be altogether absent.

Crepitation may further occur without inflammation. Thus in pulmonary cedema (dropsy of the lung) and capillary hæmorrhage in which liquids are effused in the smaller bronchial tubes and air sacs a crepitation is sometimes heard.

A modified crepitation (**dry crepitant rale** of Delafond) is usually heard over an emphysematous lung. The noise in this case has been compared to that induced by handling a sheet of paper.

The **subcrepitant rale** is another modification holding a place intermediate between the crepitant and the mucous râles. It has been likened to the sound of a moderate effervescence in beer or other liquid. It is referable to the presence of mucus in the smaller bronchial tubes and indicates bronchitis or broncho-pneumonia.

Still other sounds are heard in diseased conditions of the pleuræ. These are **friction sound**, **creaking**, **metallic tinkling**, and **gurgling** or **splashing**.

A **friction sound** is heard in the early stages of pleurisy and is caused by the dryness of the pleural surfaces from the absence of the halitus or vapor which normally moistens them, or by the deposition of layers of lymph by which the surfaces are rendered rough and uneven. An approximate sound may be observed by placing the palm of the left hand on the right ear and drawing a finger of the right hand softly over its back. The sound is quick and jerking, one or a few jerks only being heard with each inspira-

tion as the act is cut short on account of the pain attending the friction. It is rarely heard in expiration. It is chiefly heard at the lowest part of the chest where the lungs have the greatest freedom of movement. The thinness of the walls of the chest above the breast bone in cattle and dogs permits the friction sound to be heard more distinctly than in the horse. After the lapse of twelve, twenty-four or forty-eight hours the friction sound disappears, the surfaces of the pleuræ being separated by the liquid effusion, but it may reappear when the fluid is absorbed in the process of recovery. Sometimes the friction is further manifested by vibration of the walls of the chest perceptible to the touch.

The **creaking sound**, as from the bending of a piece of strong leather is caused by the movement of a thick and solid false membrane binding the lungs to the side of the chest. This is often confounded with crepitation.

Metallic tinkling is only heard when liquid and gas both exist in the pleural sac and is due to the falling of a drop from the shreds of false membrane above into the fluid contents below. The sound is somewhat like the falling of drops in a closed cask half full of water, or it may be fairly exemplified by placing the palm of the left hand flat on the right ear and striking the back of the hand smartly with the middle finger of the right. The sound is chiefly heard after the patient has changed its position and especially after rising. The explanation of this is that in the recumbent position the liquid changes its place and bathes parts which in standing are surrounded by gaseous products only. Drops accordingly fall into the liquid for some time with diminishing rapidity until they cease altogether. Other explanations of the sound but which less frequently exist are: the ascent of a bubble through the liquid and its bursting on the surface; and the sudden recoil of air from one wall of the pleural cavity to the other as the result of movement or sound generated in the deeper seated solid structures.

A **gurgling** or **splashing** sound is equally indicative of the presence of fluid and gas in the pleural sac. It is almost never
" heard unless after a sudden movement on the part of the patient causing considerable commotion in the contained liquid. Gurgling sounds transmitted from the abdomen are too often mistaken for this. In small animals with hydro-pneumo-thorax a quick shaking of the patient will develop it.

Auscultation of the cough is sometimes valuable, though more difficult and less satisfactory in the lower animals than in man, chiefly because of the extensive movement of the ribs in the former. As conveyed through a healthy lung to the ear applied on the side of the chest, the sound is short, dull and indistinct. When the lung is more solid from hepatisation, pleural exudation or other cause, or when the bronchi are dilated, the sound is loud and strong. The extent over which it may be heard thus strikingly agrees with the area of lung in state of consolidation. When a considerable cavity or canal communicates with a bronchial tube and extends to near the surface of the lung the sound is loud and ringing. The note is especially clear and metallic when such a cavity opens into the bronchus by a narrow orifice; an apt illustration of this noise may be obtained by coughing into a narrow necked vessel.

The results obtained by auscultation should be confirmed by percussion before arriving at any definite conclusion as to the state of the chest. Consolidated lung tissue is a much better conductor of sound than the healthy, and sounds conveyed through this may be heard at a considerable distance from their point of origin. Thus the heart sounds are frequently heard over any part of the right side of the chest, and crepitation and other sounds may be heard in the center of a hepatized portion. On all such occasions the dull sound elicited on percussion, will not fail to correct the fallacy.

PALPATION. TOUCH.

This is chiefly useful in cases of pleurisy. As already noticed the vibration of the chest walls which accompanies the early friction sound is sometimes perceptible by the hand applied on the side of the chest. Pressing firmly in the intercostal spaces at the affected part invariably causes wincing and, in cattle, grunting. Pinching the back in inflammatory chest diseases in cattle, but especially in pleurisy, has a similar effect.

MENSURATION.

Measurement of the chest gives less reliable results in the lower animals than in man. A cord four feet long should have one end

placed on a definite point on the withers, and not removed until both sides have been examined. It should be first carried down to a point in the middle of the breast bone and the distance marked by a knot ; a comparison may be made by carrying to same point over the opposite side. It should next be carried successively to the lower end of the eighth rib on the two sides, and the difference marked, and lastly, from the lower end of the third rib to the lower end of the eighth. These measurements should be made at one stage of the respiratory act, say when the chest is fully dilated, and similar measurements when the chest is collapsed, to ascertain any difference in the expansion of the two sides of the chest. In the smaller animals, any difference in the expansion of the two sides may be observed by inspection only, the practitioner standing directly behind the animal and watching the movements of the two sides from this standpoint.

A permanent dilatation of one side may be seen in cases of water in the chest confined to one side, and particularly if of some standing. Complete hepatisation of one lung gives a similar result. The intercostal spaces are observed to be wider than usual in such cases, and the movements of the opposite side of the chest are much more extensive than of the affected one.

A collapse with limited movement of one side is an accompaniment of chronic disease of the lung, with wasting of its substance as in the case of tubercular deposit.

DISEASES OF THE LUNGS.

Divisions of lung diseases. Bronchitis, pneumonia, pleurisy, their results, nervous disorders, asthma, hiccough. Œdema. Emphysema. Morbid growths, neoplasms. Infectious and parasitic diseases.

Inflammatory diseases of the respiratory organs situated within the chest may be divided into : inflammation of the air tubes within the substance of the lungs—*bronchitis* :—inflammation of the spongy tissue of the lung—*pneumonia* :—inflammation of the covering of the lungs and lining serous membrane of the chest—*pleurisy* :—and complicated cases in which two or more of these conditions coexist. Besides inflammatory diseases there are the various permanent morbid results of these affections, such as consolidation of lung from exuded products becoming organized ; collapse (compression) of lung from organization and contraction of false membranes ; thickening or dilatation of bronchial tubes as a result of bronchitis ; also nervous affections, such as asthma and hiccough ; morbid alterations in the lung tissue independently of inflammation, as pulmonary or pleural œdema and emphysema ; specific morbid deposits, as tubercles, glander nodes, cancer, melanosis, etc., and morbid states, due to parasites, as in the verminous affections of cattle, sheep, etc.

BRONCHITIS.

Relation to other maladies of the air passages. Horse. Causes: susceptibility, heat, cold, sudden changes, thick coat, rebreathed air, on shipboard, in zoological gardens, in close stables, in navies, organic matter in expired air, water vapor in expired air, effect on the air and bacteria. Ingesta in bronchia. Medicinal liquids in bronchia in horses and cattle. Exposed locations. Clipping. Smoke and gaseous irritants. Symptoms, in mild cases, in severe: fever, cough in dry stage, after secretion, auscultatory sounds, percussion, discharge, watery, glairy, frothy, later milky, flocculent, purulent. Convalescence. Capillary and pseudomembranous form. Intensity of symptoms, labored breathing, dyspnoea, violent cough, pinched countenance, dark mucosæ, perspirations, palpitations, asphyxia. Course, duration. Termination, difficult expectoration, blocking of bronchia, pneumonia, bowel susceptibility, skin congestion, laminitis. Chronic condition. Lesions, congestion and contents of bronchia, soft, thick, friable mucosa, absence of vascular ramification, tenacious mucus, false membranes. Collapse, atelectasis, splenisation, emphysema, bronchiectasis. Treatment, in mild cases, in severe, hygienic, steaming, sulphur dioxide, derivatives, guarded laxative, neutral salts, calmatives, expectorants, alkalies, stimulant, oxygen, peroxide of hydrogen, iodide of potassium. Diet. In advanced stages tonics.

Definition. Inflammation of the mucous membrane which lines the bronchia. It is the counterpart of *coryza* and *laryngitis* being but the inflammation of another portion of the same mucous membrane which lines the whole respiratory track. That portion of this mucous membrane which lines the trachea is rarely the exclusive seat of inflammation, so that in case of its being implicated we do not speak of the case as one of *tracheitis* but as *laryngitis* or *bronchitis*, according as the throat or bronchia form the seat of active inflammatory action.

The bronchial mucous membrane is often inflamed in influenza, strangles, contagious pleuro-pneumonia of cattle, distemper in dogs, and parasitic diseases of the lungs, but the following remarks will be confined to the simple inflammatory affection. It appears as an *acute* and a *chronic* affection.

HORSE. ACUTE CATARRHAL BRONCHITIS.

This is more frequent in the horse than in other animals, and especially so in young animals when newly stabled or put in training.

Causes. These are the same as those of *catarrh* and *sore throat*. It is but the continuation of the same mucous membrane which is affected in all alike, and the same atmospheric changes, hot stables, noxious inhalations and exposures to cold and wet, will induce this disease rather than the others, when the bronchial mucous membrane is more predisposed. Bronchitis often supervenes upon sore throat, by the extension of the inflammation downward into the chest.

As in the other affections of the respiratory tract, microbes bear an important rôle in the causation and maintenance of the inflammation. These may be specific pathogenic organisms which determine the disease whenever they are implanted in a susceptible subject, but most commonly they are mere saprophytes living habitually, in health, on the surface of the respiratory mucosa, and only proving injurious when that membrane has been injured or debilitated, or when the general tone of the system has been lowered by other conditions. As the air passages are always widely dilated, in contrast with other mucosæ which are closed by sphincters, and as the germ-laden outer air is inhaled with every breath, the floating organisms are of necessity carried in, to attach themselves to the moist surface of the canal. Though this is counteracted by the tendency of the inhaled microbes to strike against and adhere to the moist mucosa in nose, pharynx and larynx, yet a few constantly escape and reach the tubules within the lungs. These tend to reach the bronchia by gravitation. When they have reached the bronchia they are still opposed by the ever active ciliated epithélium which tend to sweep them upward and outward, until the cilia have become paralyzed and inactive through the supervention of inflammation. There is the further antagonism of the epithelial cells themselves which exercise their selective powers in excluding the microbes from the mucosa, and in destroying their vitality if they should succeed in making a partial entrance. This last influence is particularly strong in the trachea, bronchi and larger bronchia where the epithelial coating is very thick and abundant, and the resistance lessens by degrees as we approach to the smaller bronchia and the terminal or capillary tubes in which the epithelial layer becomes very thin and permeable. A successful invasion by the microbes is favored by the inhalation of

food materials during rapid breathing, in the deep inspiration following a cough, in paralysis of the larynx or pharynx, in forcible drenching, and above all in giving liquids through the nose. Gamaléia found that an irritant tracheal injection, followed in an hour by staphylococci and streptococci caused intense bronchitis in twenty-four hours.

The glands of the bronchial mucosa must also be reckoned with. These are much more abundant in the capillary bronchia than in the larger tubes. Their secretions are carried outward by the cilia and with them invading microbes tend to be washed out. But if these microbes have once gained access to the interior of the gland-ducts, through the suspension of the secretion or by reason of their own growth and increase, in the contained culture-fluid or in the epithelial lining, they have thereby secured a position of vantage from which they are less likely to be dislodged by ciliary action or by copious secretion. From these recesses accordingly they extend into the substance of the mucosa, extending the inflammation in both breadth and depth.

A similar advantage attends the microbes in case of abrasions or minute ulcers resulting from caustic or otherwise irritating gases or solids inhaled. The cilia having been removed and the phagocytic power of the tissue cells suspended or impaired, the injurious micro-organisms can work unimpeded.

Bronchiectasis and other old-standing injuries act in a similar way, robbing the mucosa of its defensive powers.

But this recognition of a causative microbial invasion must not lead us to close our eyes to many important predisposing and accessory factors without which in the great majority of cases the micro-organisms would remain harmless.

A northern or southern temperate zone, with cold, wet seasons, and all localities in which cold and damp coincide, are especially productive of bronchitis. High altitudes which are habitually cloudy (fogs) are similarly objectionable, but, in the absence of fogs, the clear, dry mountain air is beneficial. Damp air and cold wind is a fatal combination, and a low dewpoint is the main condition favoring bronchitis. Late autumn and early spring with their sudden and extreme changes of temperature and air moisture are the great periods of acute bronchitis, while the cold, dry zero air of midwinter, in our northern states, in the centre of the continent, is comparatively little injurious.

Chilling of the surface by exposure to cold drenching rains, is a frequent cause, by reason of the intimate sympathy existing between the skin and the mucous membrane. For the same reason certain conditions of the skin will predispose, thus a long, thick coat which keeps the animal constantly drenched with sweat and the skin relaxed and sensitive. Williams draws attention to the frequency and severity of bronchitis in both horses and cattle conveyed by sea during stormy weather, and especially when the hatches had to be fastened down. Such an experience combines in one the evils of overheated stall, a sudden transition often to extreme cold, a lowering of the vitality of the whole system by the circulation of non-aerated blood, systemic poisoning by the retention of the waste organic products that would otherwise have been eliminated, and the special weakening of the lung tissue by congestion of the whole pulmonic circulation.

But the development of bronchitis and broncho-pneumonia is the least fatal result. The statistics of our European cattle traffic are rich in the examples of absolute suffocation of cargoes in transit to Europe. The following from Report of U. S. Treasury Cattle Commission is illustrative :

“ Dr. Thayer reports the case of a steamer from Boston to Liverpool, with 400 cattle on board, which encountered a storm and came through it with only one animal surviving. Mr. Toffey, of Jersey City, lost 30 head out of a cargo of 300 by suffocation in 1880. This happened, he informs us, on a calm sea on a southern route with a temperature about 90° F., and the wind astern and light so as just to keep pace with the ship. The air on board the ship became perfectly stagnant, and there was no means of establishing an artificial current. A still more disastrous experience befell the steamer *Thanemore*, Captain Sibthorp, of the William Johnson & Co. line. This vessel left Baltimore with 565 cattle on board, of which 228 perished by suffocation before she reached Cape Henry.”

Among animals that survive such treatment the susceptibility to lung disease including even the contagious forms like tuberculosis is enormously enhanced.

EFFECTS OF MODERATELY VITIATED AIR.

“ When air only moderately vitiated is breathed continuously for a greater length of time the results are still very injurious,

and in the front rank of diseases so caused stand pulmonary consumption, and other destructive affections of the lungs. Perhaps no better example of this can be given than that of the monkey houses of the Zoological Gardens of London and Paris. While these houses were small and ill-ventilated the monkeys died in large numbers from pulmonary consumption, but after they had been enlarged and better ventilated the mortality from this cause nearly ceased. (Arnott.)”

“Town dairy cows which are packed in close ill-ventilated buildings and never allowed to go out are very subject to consumption, while horses kept in no better conditions, but spending nearly half their time in the open air, rarely have phthisis. (With lung plague it will be remembered that the out-door exercise and mingling of herds leads to an increase of the mortality.) Horses newly stabled suffer severely from diseases of the lungs. The same holds true of human beings. A long list of careful observers have noticed the essential connection of lack of ventilation and pulmonary consumption. Baudelacque, Carmichael, Arnott, Lepelletier, Allison, Sir James Clark, Toynbee, Guy, Greenhow, Sir Alexander Armstrong, Parks and Aitken have especially insisted upon consumption being a sequence of a lack of ventilation. Dr. Cormac indeed held that consumption is originated by rebreathed air.

“The notorious prevalence of consumption in sailors has been directly traced to the impure air in which they sleep, and an extensive outbreak of lung disease (not tubercular), leading to destruction of lung tissue, in the English Mediterranean squadron in 1860, was clearly traced by Dr. Bryson to the contamination of the air. In a nursery hospital at Dublin, with entire neglect of ventilation, 2,944 children died in four years, whereas after the ventilation had been improved only 279 died in the same length of time.”

“Parkes (Practical Hygiene) says :

“‘But not only phthisis may be reasonably considered to have one of its modes of origin in the breathing of an atmosphere contaminated by respiration, but other lung diseases, bronchitis and pneumonia, appear also to be more common in such circumstances. Both among seamen and civilians working in confined, close rooms, who are otherwise so differently circumstanced, we find an excess of the acute lung affection.’”

“ In this connection, the statement of the air breathed by an ox per hour and that supplied him on board a ship with insufficient ventilation or none may be instructive. The ox takes in with each breath about 5 liters of air. This is at the rate of 50 liters per minute, or 3,000 per hour = 105.9 cubic feet. This amount of air is therefore rendered all but irrespirable by each animal in the course of an hour. And this, be it noted, is by breathing alone, and makes no account of the contamination by perspiration in the overheated hold, and by the emanations from the accumulating excrement.”

“ On board the steamers we have found the space allotted to each bullock to vary from 150 to 240 cubic feet. On the steamship “ Holland,” loaded at New York, August 21, 1881, we found the stalls amidships allowed the full space of 240 cubic feet per head. In the bow where there was less height between the decks the space was considerably less. On the lower deck, where 129 cattle were accommodated, the space allowed each was 217.4 cubic feet. The port-holes in the upper deck were nine inches in diameter and there was one for each pair of stalls — central and lateral or for eight oxen. These being well above the water line would be available for ventilation in ordinary weather. The port-holes in the lower deck, similarly arranged, were about two feet above the water line, and consequently not available for ventilation, save in exceptionally calm weather. The temperature on the main deck of this ship (between the outer and main deck), when only half the cattle had been loaded, was in the neighborhood of 90° although she was lying in the center of the North River with port-holes and hatches open, and a fresh breeze blowing from the north.”

“ On the ‘ Assyrian Monarch ’ the space per head was only 192 cubic feet, but this ship was supplied with a ventilating fan or blower capable of delivering over 50,000 cubic feet of fresh air per hour, so that her ventilation was abundantly provided for. In some smaller ships we found the space per head to exceed little, if at all, 150 cubic feet. In these, accordingly, a single hour without any change of air would threaten the life of every animal on board, and two hours would endanger those for which even the larger space is provided. It is true that such absolute seclusion is rarely required, and that a certain amount of aerial

diffusion is always going on through imperfectly closed hatches, companion ways, and ventilators, yet, that these are often insufficient has been amply shown by such losses as are reported above, as well as by the bronchitis and tuberculosis which Drs. Whitney, Lyman, and Williams have found in the lungs of American animals arriving in England."

"ORGANIC MATTER IN EXPIRED AIR."

"The decomposing organic matter given off by the lungs and skin is probably the most injurious of the animal excreta, when allowed to act on the system for a length of time. This exhaled organic matter is early recognized in the air by chemical tests, or by the putrid odor evolved when cotton wool, that has been breathed through, is left to soak in otherwise pure water at a temperature of 70° to 80° Fahrenheit. The experiments of Gavarrat and Hammond, in which expired air had its carbonic acid and water vapor removed, leaving only the organic matter, showed that the latter was highly deleterious. Hammond found that a mouse died in forty-five minutes in such an atmosphere. It has also been again and again demonstrated that air containing a given amount of carbonic acid as the result of respiration is far more poisonous than air which contains the same amount of carbonic acid as a product of combustion."

"WATER VAPOR IN EXPIRED AIR."

"The amount of water vapor given off by the lungs varies greatly according as the air is already more or less saturated with water. As the air in the stalls between decks is always saturated with water vapor, we may take the very lowest estimate for each animal, namely, 60 ounces in 24 hours, which for a cargo of 200 head would amount to over 93 gallons. And this is in addition to the exhalations from the skin and the bowel and kidney excretions. The air between decks is therefore constantly saturated with moisture which condenses and runs down in streams on every solid object. Among the ill effects of this saturation may be noted :"

"First. The saturation of the air with water vapor increases the exhalation of carbon dioxide from the lungs. This effect on

the excretion of carbonic acid is usually so great as to counter-balance the tendency of warm air to reduce the production of this acid. This saturation, therefore, with water, increases the danger of suffocation by the accumulation of the irrespirable carbon dioxide in the ship, unless the air is being constantly removed.

"Second. The excess of moisture in the warm atmosphere hastens the decomposition of the organic matter derived from the lungs, skin, and manure. Sir Alexander Armstrong, head of the medical department of the British Navy, says : 'There can be no more fertile source of disease among seamen, or, indeed, other persons, than the constant inhalation of a moist atmosphere, whether sleeping or waking ; but particularly is this influence injurious when the moisture exists between a ship's decks, where it may be at the same time more or less impure, and hot or cold, according to circumstances.' It has become an aphorism with sanitarians that 'a damp ship is an unhealthy ship,' and many instances are adduced in which a sufficient renewal of the air between decks, with or without stoves to dry it, has transformed a naval pest-house into a salubrious vessel.

"All such considerations must emphasize the demand for such a constant renewal of air between decks on steamers carrying cattle, as shall serve to obviate all those conditions of ill-health, with congestion and inflammation of the lungs, as have proved in the past a serious drawback to our foreign cattle-trade. To accomplish this and at once remove from between decks the excess of carbon dioxide, of decomposing organic matter, and of humidity, and to furnish air approaching in purity and dryness that of the atmosphere outside, we can conceive of nothing more simple and effective than thorough ventilation by fan or heat extraction, as referred to below." Report of the United States Treasury Cattle Commission, 1882.

The above quotations were written with special reference to cattle, but the author reproduces them here as in principle applicable to horses as well.

In both horses and cattle treated as above it is common to find ingesta in the bronchia drawn in during the violent paroxysms of coughing. Here we have a direct mechanical irritant and a means of septic infection, highly calculated to induce unhealthy broncho-pneumonia. Williams quotes the case of a horse in

which vomition was caused by an over dose of aconite, and a portion of the food entered the bronchi.

In this connection must be named the introduction into the bronchia of liquids forcibly administered to horses and cattle. In the horse the length of the soft palate enables him to hold liquids in the mouth during his pleasure, and among the expedients adopted to compel him to swallow are the very dangerous ones of holding the nostrils, and of pouring the liquid through the nose. When the nostrils are held the urgent demand for air leads to attempts to breath through the mouth, and whether he succeeds in this or not, the usual result is the drawing of a portion of the liquid into the lungs. When it is poured through the nose the animal cannot protect himself except by rapid gulping, and as he must breathe, a portion of the liquid is usually drawn into the lungs. Any irritant taken in this way will develop bronchitis, and some bland agents like melted lard are almost equally injurious. Cattle having a short palate can scarcely resist swallowing liquids that are poured into the mouth, but a cough with the succeeding quick inspiration will almost certainly draw a portion into the bronchia. To return to the influence of cold, exposed situations which receive the full force of cold winds, those from the north and west on the Atlantic slope, are especially conducive to bronchitis. Exposure of newly clipped animals to stand without protection in winter or early spring, has the same tendency. Finally the inhalation of smoke or of heated and irritant gases and vapors, as in a burning building, is an effective factor.

Poisoning by mercury or alkalies determines catarrhal bronchitis.

Symptoms. In its *mildest form* catarrhal bronchitis is a transient illness with some dullness, impaired appetite, hot, dry mouth, redness of the visible mucous membranes, a moderately strong, resonant cough, attended with slight fever, slight rise of temperature, accelerated breathing and pulse, and mucous discharge from the nose. Such an attack passes over in a few days and without any medicinal treatment if ordinary precautions are taken to avoid a repetition of its causes.

In *severe cases* the symptoms are more intense from the first, microbial invasion and absorption of toxins causing more general

disorder. Besides the dullness and inappetence, hot, dry mouth, generally increased temperature of the body (102° to 104° F.), accelerated and labored breathing with double lifting of the flank in expiration, and other manifestations of fever, there are more specific symptoms. The cough is dry, hard, painful, often paroxysmal, and appears as if it came from the very depth of the chest. A strong, harsh, blowing bronchial sound is heard over the lower end of the trachea and the upper border of the middle third of the chest, just behind the shoulder. Percussion detects no change from the natural resonance of the chest, nor auscultation any crepitating sound. Pressure in the intercostal spaces causes no suffering. The expired air feels hot. The pulse though accelerated is moderately soft and sometimes even weak, a condition which marks inflammations of mucous membranes as contrasted with those of the serous. The mucous membrane of the nose has a dark red hue, especially when the inflammation extends to the smaller ramifications of the bronchial tubes so as to impair the æration of the blood. In the same state there is excessive dullness and prostration because of the supply of partially venous blood to the brain. The head is held low, the nose often supported upon the manger, and the eye-lids are semi-closed and injected.

From the second to the fourth day a free exudation takes place from the surface of the mucous membrane, and the symptoms are materially changed. The cough becomes more frequent but softer, looser, and attended with a rattle, heard in both inspiration and expiration, from the air passing through the abundant mucous secretion. The cooing or tubal sound heard at the lower end of the windpipe and behind the shoulder has now given place to a *mucous râle*. A nasal discharge appears at first watery, thin, of a whitish, glairy froth, but soon becoming more opaque, white, milky and flocculent and having little tendency to stick to the nostrils. This is often expelled with sneezing and accompanied by movement of the jaws. With the access of free secretion there is a great mitigation of the fever and the other distressing symptoms, and, if no relapse nor complication supervenes, recovery may be complete in a fortnight or three weeks from the onset.

From this time all the febrile symptoms decline and disappear,

appetite and liveliness return, the discharge rapidly diminishes and finally disappears, when the patient may be said to have completely recovered.

Capillary and pseudo-membranous bronchitis are described by Reynal as occurring in young horses (three to seven years,) recently brought into the army and subjected to the hot and close stables in some of the French barracks. It began as ordinary bronchitis, which in place of tending to recovery, propagated itself to the most minute bronchial ramifications, and was frequently complicated by the formation of false membranes. The signs of its accession are an extreme intensity of the general symptoms, the rapid, labored difficult breathing, accompanied by convulsive action of the pectoral and abdominal muscles; the frequent painful, suffocating and abortive cough, which violently shakes the whole body; the extended head, open mouth, distorted nostrils, reddish brown protruding eyes; the pinched, haggard features, and the frothy mucous, nasal discharge striated with blood, and later interspersed with shreds of false membrane similar to those existing in croup. In connection with these are the symptoms of extreme oppression, partial sweats, tumultuous action of the heart and small, weak, rapid pulse. Death resulted from suffocation during a paroxysm of coughing.

Capillary and pseudo-membraneous bronchitis are especially liable to be associated with pathogenic microbes, which not only take occasion to attack the mucosa which is already the seat of debility or structural lesion, but which may cause secondary infections (endocarditis, pericarditis, nephritis), and inoculation of the Guinea pig with the exudate has caused death by rapidly advancing septicæmia (Lorge).

Course. Duration. Termination. Catarrhal bronchitis is not usually fatal, except in very young or old or worn out animals, or unless it assumes the *capillary* form or is complicated by pneumonia, pulmonary abscess or by metastasis to the bowels or feet. In the mildest cases health is re-established in three or four days, and in the severe, about the twelfth, fifteenth or twentieth day. In old and debilitated animals in which pure bronchitis proves fatal, the abundant effusion into the bronchial tubes, the influence of gravitation retaining this in the smaller tubes, the palsy of the cilia which normally carry it outward, and

the want of power to expectorate by coughing, usually bring about suffocation. This is favored by the nonærated state of the blood, which rapidly prostrates the already weakened nervous centres. The supervention of pneumonia will be marked by a new class of symptoms, especially labored breathing, dullness on percussion and crepitation on auscultation. The susceptibility of the bowels is so great in some cases of bronchitis, particularly in those associated with a low fever, that superpurgation, enteritis and death may result from the smallest dose of laxative medicine; the author has seen a fatal result from the administration of the two drachms of aloes in a case of this kind. In such circumstances the skin usually participates in an equal degree, and though the superpurgation be checked, an extension of this disease to the feet may still prove fatal or induce such changes of structure as to leave the animal practically worthless. In old animals or after repeated severe attacks of bronchitis it may merge into the *chronic* form. *Thick wind* is a frequent sequel of severe cases from thickening or dilatation of the bronchial tubes, from collapse of the lung or from emphysema.

Post-Mortem Appearances. In the bodies of animals that have died of bronchitis the air-passages within the lungs are filled with a white or greenish yellow mucus. If this is washed from the tubes by a stream of water, the mucous membrane is often found to be injected, studded more or less profusely with red points or with branching red lines, and with petechiæ, and it is further softened, sometimes thickened and friable. When, however, the bronchitis has been attended by a free purulent expectoration the mucous membrane may, when washed, show no perceptible alteration from the healthy standard as examined by the naked eye.

In the *capillary form* the blocking up of the smaller tubes by a tenacious frothy mucus, and by the false membranes which form complete casts of many of the tubes and the partial consolidation (collapse) of circumscribed pyriform masses of the lung tissue with which such tubes communicate, form the chief features on examination after death.

This state of *consolidation* or *collapse* of lung is frequently seen in simple bronchitis as well. It is then due to the blocking up of one or more bronchia by plugs of tenacious mucous which act

as valves, preventing the entrance of air, though they may permit of its easy passage outward. This state of lung differs materially from the consolidation due to inflamed lung tissue (hepatisation). When cut it does not present the granular appearance of the latter, caused by the exudation into the minute air cells, but the cut surface has an uniform homogeneous aspect, aptly likened by Lænnec to *muscular flesh* (splenisation). Mendelson, Traube and Gairdner have induced artificial collapse of the lung by introducing foreign bodies into the bronchia of animals.

Emphysema of the margins of the lung is a frequent concomitant of *collapse*. The cause is plain. The portion of lung, the subject of collapse, emptied of its air, does not occupy a tithe of the space it would normally fill. The rest of the lung tissue expands unduly, to fill out the vacated portion of chest, and the cells become over-distended and ruptured. The emphysematous lung is known by its lighter color, by its irregular bulging surface, by the subsidence of these elevations when pricked with a needle, and by a more marked crepitation when pressed. When the cells have burst and the air escaped into the areolar tissue between the lobes, it appears as dark lines circumscribing small portions of pulmonary tissue, and collapsing when pricked.

Treatment. Some general principles rule in treating acute bronchitis. These may be suggested as follows:

Rest is all-important to allow recuperation, restoration of vigor, and the removal of the exhaustion, low condition and debility, and the hurried breathing, which have acted as prominent causative factors.

Pure air at a genial temperature constantly maintained, guards against further irritation and soothes the inflamed passages. Seek a sheltered box with sunny exposure.

Moist warm air is especially soothing, as moist cold air is particularly injurious.

In great dyspnoea with irritable contracted bronchia inhalation of *nitrite of amyle*, *fumes of burning saltpeter paper*, *iodide of ethyl* or chloroform will relieve, giving easy breathing and better æration of the blood. In the absence of these, *hyoscyamus*, *belladonna*, *stramonium*, *tobacco*, or *lobelia* may be tried.

Sedative expectorants are of great use in the early, irritable, dry condition of the mucosa, and may be found in salts of the

fixed alkalies—soda and potash—particularly in *iodides* and *bromides*, in *ipêcacuanha*, and, to a less extent, for herbivora, in the salts of *antimony*.

Stimulant expectorants encrease secretion and give tone to the mucosa, and are especially valuable in cases in which the bronchitis is already well established, during convalescence, with weak heart, and in the chronic cases. These include inhalations of *ammonia* or *alcohol*, *strychnine*, *squills*, *senega*, all *aromatic volatile oils*, *oleoresins* and *balsams*.

Antiseptic expectorants are especially needed when the sputa is delayed and decomposed in the tubes, causing a foul smelling breath and discharge. They include *carbolic acid*, *creosote*, *creolin*, *cresyl*, *terpin*, *terpene*, *terpinol*, *terebene*, *oil of turpentine*, *oil of tar*, *menthol*, *eucalyptol*, etc. They may be administered, diluted, as intratracheal injections, by inhalation with steam, or in an atomized condition. Like stimulating expectorants, these are of great value in many chronic cases.

For stimulating the respiratory centres directly *strychnia* and *ammonia* are especially indicated, while to quiet their irritability, *opium*, *bromides*, *chloral*, and *chloroform* may be used. *Opium* is usually objectionable in the early stages as calculated to arrest and prevent secretion, but in combination with an expectorant like *ipêcacuanha* it forms an ideal soothing expectorant.

The mildest cases will recover of themselves, especially if care is taken to protect the patients against cold, wet, draughts of cold air, over-exertion, and other injurious causes, and to give a part of the food warm and sloppy. In severe cases treatment must be more active, but it will be borne in mind that severe depletive measures are badly endured. Bleeding dangerously increases the already existing weakness and prostration, without affording any corresponding advantage. It is only admissible when from the severity of the symptoms in the early stages, suffocation is threatened, or, when the brain becomes involved in disease.

Causing the patient to inhale water vapor from scalded bran or hay is to be assiduously carried on for half an hour to an hour twice or thrice daily until expectoration has been freely established and the cough and fever alike moderated. The density of the vapor must of course be apportioned to the particular case so as

to avoid any approach to suffocation. The addition of the fumes of burning sulphur will often by their astringent and antiphlogistic action on the mucous membrane, render the vaporous application more effective. A pinch put into a small piece of paper twisted at one end to prevent burning of the fingers, may be set fire to and the fumes allowed to pervade the apartment so that they can be breathed freely without inducing cough. A mustard poultice to the neck and sides of the chest should also be applied and kept on an hour or until effusion into the skin is well marked by thickening of its substance. Injections of warm water should be given alike to check or obviate shivering, to equalize the general temperature and to solicit the action of the bowels. In sporadic cases with active fever and full strong pulse a laxative dose of aloes (3 to 4 drachms) may be given, but if with a low fever, and during the prevalence of influenza, not more than half the dose should be given, or enemata alone may be relied upon. As soon as the medicine has set or at once if it is withheld, neutral salts may be given (Liquor Ammoniaë Acetatis 2 oz. or Sweet Spirits of Nitre 1 oz. or nitrate of potass $\frac{1}{2}$ oz. combined with 10 drops tincture of aconite repeated twice or thrice daily). If the cough is troublesome and secretion long in being established, expectorants may be used (oxymel of squill 3 oz., powdered squill $\frac{1}{2}$ oz. or liquorice 1 drachm) with half the doses of the neutral salts. Dover's powder 2 drs., chloral, conium, lobelia, etc., will check the cough.

In the early stages to hasten expectoration such preparations as the following may be given :

Recipe: Potas. Bicarb. 2 ounces; Ammon. Carb. 2 ounces; Digitalis Pulv. 2 drams. Mix. Divide into eight powders: give one every four hours.

Recipe: Ammon. Murias, 2 ounces: Choral Hydrat. 1 ounce; Tinct. Hyoscyam. 2 fluid ounces; Aqua 8 fluid ounces. Mix. Give two tablespoonfuls every four hours.

Apomorphia, tartar emetic, turpentine, glyco-heroin, quaiacum or benzoin may be employed, or even pilocarpin, care being taken not to increase prostration unduly. Compressed air, oxygen and peroxide of hydrogen will sometimes relieve.

Intratracheal injections of solution of silver nitrate ($\frac{1}{2}$ —1:100) (Levi); of oil of turpentine and olive oil, $1\frac{1}{2}$ dram each; or of

iodine 15 grains, potassium iodide, 1 dram, water 6½ ounces, (Dieckerhoff) are often useful. They sometimes prove too irritating, and have even led to gangrene and sloughing.

With the advent of expectoration, or earlier, iodide of potassium in one drachm dose, thrice a day will do much to obviate glandular and other enlargement which would tend to develop *roaring*.

The diet should be laxative, non-stimulating and somewhat spare. Mashs of wheat bran, boiled linseed or boiled barley; roots such as turnips, carrots, beets; in summer a limited supply of fresh grass, with a little hay at any time and that scalded, may indicate the nature of the aliments to be used. As a beverage chilled fresh water or linseed tea may be supplied *ad libitum*.

Should the nasal discharge manifest no disposition to cease at the end of 15 or 20 days, as will sometimes happen in young horses, stimulants and tonics must be employed. Gentian (4 drachms), Sulphate of iron (2 drachms), Arsenious acid (5 to 10 grains) or nux vomica (1 scruple) may be given daily as ball, electuary or powder. A full and nutritive diet should at the same time be allowed, and open air exercise enjoined.

CHRONIC BRONCHITIS IN THE HORSE. BRONCHIAL CATARRH.

As Sequel of Acute : as result of unhygienic environment, diet and usage. Symptoms, breathing accelerated, double expiratory act, short breath, cough husky, or paroxysmal, excited by cold air, water, discharge white flocculent. Percussion, drum-like patches ; Auscultation mucous and sibilant râles. Lesions, thick mucus, pale membranes, bronchiectasis, emphysema, ulceration. Thick wind. Treatment, stimulating, tonic, derivative, medicated vapors, careful diet. Tar water.

This may be a sequel of the acute form, or it may appear at once as a catarrhal discharge from the bronchial tubes and without any very marked febrile affection, in animals debilitated by damp stables, overwork and faulty regimen and diet.

Symptoms. Respiration accelerated, and expiration effected with a double lifting of the flank ; the horse is easily blown when moderately exercised ; the cough is frequent, soft or rattling and paroxysmal—sometimes hard and deep—is excited when brought from the warm stable into the cold air, and is followed by a whitish, flocculent, purulent discharge from the nostrils, consistent but not sticky (like buttermilk). The pulse is rapid and small in volume.

Percussion manifests a healthy resonance over the greater part or all of the chest, a drum-like sound over given areas, auscultation over its median part detects a bronchial rattle and in most cases a dry sibilant râle.

After death the bronchial tubes are found to contain an abundance of thick mucus, though abnormal redness of the mucous membrane is by no means a necessary condition. There is always more or less dilatation of the bronchial tubes especially at their points of subdivision where they are often twice their healthy calibre, and an emphysematous state of the lungs is equally constant. The walls are often greatly thickened. Delafond and Rodet have noted minute ulcers on the bronchial mucous membrane and Reynal miliary abscesses and grayish and white indurations of the lung tissue and bronchial glands which may have been glanderous. Nodular indurations result from peribronchitis

as follows: Inflammation extends outward from the mucosa into the outer coat; this is thickened by exudation and cell proliferation, especially in the nonstriated muscular fibres; at intervals this degenerates into pus, forming minute abscesses of the size of a lentil to a pea, each with a thick outer coat; the intervening portions of the exudate are more or less absorbed, so as to leave a series of peribronchial nodules (peribronchitis nodosa). These are differentiated from glander nodules, by being all of one age; by their pallor in contrast with the reddish color of their section in glanders; and by the presence of a few drops of creamy pus in the centre of each.

It will be observed that the symptoms and lesions closely resemble those of heaves, and unless early and successfully treated, into this it gradually merges. The chief distinguishing symptoms are the abundance and nature of the discharge, the fetor of the breath, and the presence of the mucous râle in the chest. It is one of the conditions known by the horsemen's expression "*thick wind*."

Treatment. Like its type (dilatation of the bronchia, bronchiectasis) in man this disease obstinately resists treatment. In our efforts to cure it the same general principles must be followed as in acute bronchitis, with this grand qualification that the general aim must be to stimulate and support. Stimulating liniments may be repeatedly applied along the course of the trachea and on the sides of the chest. An equable temperature is desirable and a dry building. Water vapor medicated with various astringents and antiseptics (creosote, carbolic acid, turpentine, tar, or tar vapor) is to be commended. A course of tonic and expectorant medicine is desirable and a highly nutritious and laxative diet is imperatively demanded.

As tonics Gentian may be given daily in 4 drachm doses combined with quaiacum in doses of 2 scruples, or sal ammoniac 2 drachms. In most cases it will be advisable to add to the above or employ separately arsenious acid in doses of 5 to 10 grains combined with three times the amount of bicarbonate of soda, and given daily for a month or longer. When the cough is dry and husky iodide of potassium or chlorate of potash may be useful, while with profuse expectoration, wild cherry bark (syrup) may be resorted to.

The diet should be as for broken wind, nutritious, in small bulk, of a laxative nature, and given at least an hour before work. A moderate supply of grass, roots, bran, oats or barley may be given, but hay must be sparingly supplied and, if exclusively clover hay, dry and dusty, is better withheld. In the north of France horses with chronic bronchitis are maintained in a serviceable condition by a diet of cut straw and cut hay, well sifted to remove all dust, mixed with oats and molasses and set aside in a large cask to ferment before being given to the animals. Tar water may be the exclusive drink.

A pint of linseed, well boiled, and given daily for a length of time in succession is often of great value.

CATARRHAL BRONCHITIS IN THE OX.

Working oxen most susceptible. Causes, damp buildings, wet, cold, exposed localities. Debility, overwork, poor feeding, close, foul stables, sudden change of temperature. Previous attacks. Symptoms, mild form, and severe, fever, sneezing, cough, dry, later soft, prostration, retraction of nostril, labored breathing, hot breath, discharge, watery, milky, purulent. Duration. Complications and sequelæ. Chronic form. Respiratory disturbance, paroxysmal cough, purulent discharge, mucous and sibilant râles, emaciation. Lesions. Treatment, laxative safer, expectorant, derivative stimulant, etc., as in horse. Bronchitis in pigs and sheep.

This is less common than the same disease in the horse, though in working oxen, in which many of the same causes operate, it is frequently seen. It is not infrequent in other cattle in damp buildings, or in wet, cold exposed situations. Debility from overwork and poor feeding, often brings on the chronic form of this disease. Living out in damp nights after a hard day's work is another frequent cause. The enervating influence of the hot foul air of many cow houses conduces to it and is specially injurious if alternated with a chilling atmosphere out of doors. Previous attacks strongly predispose to future ones. Microbes fill an important role in the inflamed weakened tissues.

Symptoms. Some cases are so slight as to escape a cursory observation and subsiding in a few days leave the animal perfectly well. Others are severe and may prove dangerous.

The earlier symptoms are dullness, staring coat or shivering, and sneezing, followed by reaction with hot clammy mouth, general increase of temperature, rapid pulse, reddened nose and eyes, and suspended rumination. The more characteristic symptoms are a hard, dry hacking cough, not so resonant as in the horse, but with open mouth and protruding tongue, and soon a mucous discharge from the nose usually cleared away by the tongue almost as rapidly as formed.

If the case increases in severity, and in many cases almost from the first there is great depression, hanging head, semi-closed watery eyes, extreme movement of the nostrils, hot expired air, labored action of the flank, complete loss of appetite, constipation, fæces covered by mucus, cough very hard, painful, oc-

curring in paroxysms and easily excited by touching the larynx or trachea. This clears the passages, relieving the breathing and temporarily arresting the stridor. This is followed by a loose cough, a free discharge from the nose and a mucous or sibilant râle on auscultation. Percussion causes grunting but gives healthy resonance. The disease reaches its height on the fifth day and recovery may be almost perfect on the eighth. Its chief danger is from a complication with pneumonia or pleurisy, or from its merging into the *chronic form*.

Chronic bronchitis in the ox is characterized by a persistent disturbance of the respiration, paroxysms of coughing, a white flocculent discharge from the nose, increasing emaciation, pallor of the mucous membranes, a mucous râle over the windpipe and median part of the chest and a cooing sound over other points. If left to itself emaciation becomes extreme, the skin is harsh, inelastic, attached to the ribs and covered by vermin, and death usually ensues from diarrhœa or tuberculosis.

After death the lesions are like those seen in the horse, unless there is the complication of tuberculous or other disease of the substance of the lungs.

Treatment. Neither the general care nor the remedial treatment differs materially from that for the horse. The principle difference is in the lesser liability to superpurgation and in the preference to be given to Epsom or glauber salts over aloes as a laxative. Either saline may be given in dose of one pound combined with an ounce of ginger or other stimulants, and followed up by similar diuretics, expectorants and tonics, as in the horse.

The *chronic form* is to be treated as in the horse.

Sheep affected with bronchitis must be treated on the same general principles as the ox, only giving one-fifth the amount of the different medicaments.

CATARRHAL BRONCHITIS IN SWINE.

This is rare as the result of such climatic vicissitudes as cause the disease in other animals. Nevertheless the severe forms of laryngitis, aggravated by microbial invasion, are liable to extend to the bronchia, and again it has been traced to smut of corn.

More commonly the bronchial catarrh is caused by the *strongylus paradoxus* in the air passages, especially in young and growing animals.

Symptoms. In simple bronchitis there is early the hoarse raucous cough, merging into the loose, mucous or gurgling one, with a more or less abundant, filamentous, or muco-purulent discharge. In the form which results from smut, there is the accompaniment of irritation of the digestive organs, with profuse diarrhœa, and the fever, pulse, and general symptoms of bronchitis. In the bronchial strongylosis the general symptoms are complicated and explained by the presence of the round worms or their eggs in the expectoration. The bronchial sounds are liable to be wheezing and the chronic cases may show little obvious symptom beyond a frequent cough and a persistent low condition.

Lesions. In the form determined by smut the tracheal mucosa shows fine punctiform petechiæ and erosions. These are continued into the bronchia and, on microscopical section, the mucosa shows numerous clubshaped cells perforated by punctiform openings. These are the spores of the smut imbedded in the membrane and causing necrotic degeneration. In other respects there are the marked congestion of the mucosa, the softening of the epithelium, its desquamation, the resulting erosions, and the covering of muco-purulent matter. The walls of the bronchia and the peribronchial connective tissue may show fibroid degeneration even to sclerosis. The lower portions of the anterior and median pulmonary lobes are liable to be infiltrated, consolidated with numerous yellowish foci having each a clearer transparent outer zone.

Treatment. Inhalations of warm water vapor, sulphur fumes, volatilized ammonium chloride, carbolic acid, creolin, oil of turpentine, may be given. A laxative, followed by a course of ammonium chloride, ipecacuanha, or senega may be useful. Warm, sloppy drinks and mashes are all important. Soothing warm compresses to the chest are equally important, to be followed by active frictions with a liniment of equal parts of ammonia and oil closely covered for half an hour to prevent evaporation.

Bronchitis from smut demands especially warm water vapor inhalations, impregnated with oil of turpentine, camphor, or menthol.

SIMPLE CATARRHAL BRONCHITIS IN THE DOG.

Causes: damp kennels, cold and damp after hunting, pampering and exposure, distemper. Symptoms: fever, cough hard, later soft, discharge watery, glairy, purulent. In capillary bronchitis cough more paroxysmal painful and attended with vomiting. Disturbance of breathing, pulse, temperature. Fatality in different breeds. Treatment: laxative, expectorants, diuretics, heart tonic, calmative, water vapor, chest jacket, stimulant expectorants, stimulants, tonics. Diet.

This is common and severe. Hounds kept in damp kennels, much exposed to cold and damp after being heated in hunting, or subjected to frequent and sudden alterations of temperature are specially liable. Pampered pets kept in warm rooms, overfed and having little open air exercise, are equally subject to its attacks. It is an usual form in which distemper is manifested.

Symptoms. There is roughness of the coat or shivering and a small, hard cough, often repeated. If confined to the bronchi the cough soon becomes loose, a free discharge sets in, and with care recovery may be secured in five or six days.

If the smaller bronchial tubes are involved the symptoms are more intense and persistent. The temperature may reach 104° or 105° . To the same early symptoms succeeds a painful cough, occurring in paroxysms and sometimes followed by vomiting of a glairy mucus. There is running from the eyes and nose, and reddening of their membranes. The creature stands with his elbows turned out, his flanks heaving and his heart beating rapidly and tumultuously. In the worst cases when the inflammation has been propagated to the smallest bronchial tubes constituting *capillary bronchitis*, these symptoms are seen in the most aggravated type and the subject often dies of suffocation, or by implication of the lung tissue. Percussion and auscultation are even more applicable than in the larger animals, showing the clear resonance of the lung tissue, the tubal murmur in the early stages and the mucous rattle in the later ones. In the *capillary form* a distinct crepitation is heard like that of pneumonia but without central, flat percussion sound. Bronchitis proves most fatal to the higher bred dogs, such as King Charles spaniels, Italian greyhounds, and English terriers, and according to St. Cyr, small dogs suffer more severely than large ones.

Treatment. If the bowels are costive a tablespoonful of castor oil should be given, followed up by nauseating expectorants and

diuretics (tartar emetic, two grains, nitrate of potass, one drachm, and sugar, one drachm, mix thoroughly, divide into twelve powders and shake one on the tongue thrice daily). If the cough is very troublesome a grain of powdered digitalis may be added to each dose, or after the nasal discharge has been freely established one-half grain of opium among the tartar emetic. The opiates are of great value in controlling the paroxysms of coughing and the propagation of the disease to the smallest ramifications of the air passages, but as they check secretion they must be used with caution until a free discharge has been established. Dover's powder may be given early. In the early stages bromides, hyoscyamus or digitalis may be preferred. Muriate or carbonate of ammonia, syrup of Tolu, senega, or guaiac may follow.

The following may be used as expectorants: Tincture of anise, twenty drops, aqua ammonia, twenty drops, alcohol, one oz., water, one oz.: Dose one teaspoonful several times a day. Or, chloride of morphine, two cgrms., apomorphine, three cgrms., muriatic acid, three drops, distilled water, 150 grms., (Rossbach). Dose, a teaspoonful in soup thrice a day. Fortuna advises—eucalyptol, twenty grms., olive oil sterilized by heat, 80 grms. Dose, one cubic centimeter, subcutem once or twice a day.

In case of high fever acetanilid, $\frac{1}{4}$ grm., may be given several times at intervals of six hours.

Inhalations of water vapor with phenic acid, of terebinth, etc., the maintenance of an equable temperature, and the moist chest jacket, followed by mustard poultices to the throat and chest are not to be neglected. The diet should be simple, oatmeal or Indian corn pudding with milk, soups and the like may be allowed, but as a rule, butcher meat is to be withheld. If the patient has previously fed entirely on the latter it should now be given in very limited amount only, and qualified by an admixture of farinaceous diet.

In some cases the prostration becomes so great that the patient must be supported by tonics and stimulants (a teaspoonful each of sweet spirits of nitre and tincture of gentian, or a teaspoonful of port wine repeated twice daily).

In case of persistent discharge, iron, liquor arsenicalis, the same strength as Fowler's solution, or cod-liver oil may be used.

MICROBIAN BRONCHITIS IN DOGS.

Causes. Infectious bronchitis is a very common manifestation of distemper in dogs, but apart from this, slight bronchitis and sore throats are particularly liable to be complicated by the serious invasion of the inflamed and debilitated mucosa by microbes which have been living as saprophytes, it may be out of the body, or it may be on the pituita, mouth, fauces, tonsils, pharynx or larynx, or even on the bronchia themselves. All such conditions as operate in other animals, hurried breathing, sudden inspiration after cough, and paresis of the cilia, etc., are equally effective here in favoring the entrance of the microbes into the lower air passages, and their colonization on and even in the mucosa. Once established there the invading microorganisms adapt themselves to the new environment and more readily attack other animals, though these may not have been subjected to the same original exciting cause. Thus it may start in a dog which has been fatigued with hunting, chilled by being plunged in ice cold water, stood in a current of cold air, or which had to sleep on a cold stone or metal plate, but the first case is likely to be followed by another and another until the whole kennel suffers. The malady becomes for the time infectious and is to be distinguished from distemper largely by the constancy with which it attacks the throat and bronchia to the exclusion of other parts of the body. All its victims suffer in the same way in the air passages only, while in distemper different patients are liable to have the inflammation concentrated on different organs, one suffering especially in the eyes, another in the nose, another in the throat, the lower air passages or lungs, another in the liver, stomach or bowels, another in the nervous system, and another in the skin. Even apart from this in distemper the same dog is liable to develop in succession morbid phenomena referable to to these different parts, implying the presence of a germ which prevades the whole system and may dominate first the weakest and less resistant organ. The infectious bronchitis on the other hand, starting from invasion by saprophytic organisms, usually confines itself to the bronchial mucosa, and shows little tendency

to extend beyond the air passages and lungs even in specially severe cases.

Cases of this kind, however, may come into a kennel with the contagious quality already fully developed, as when a dog has been at a show, a coursing meeting, a hunt or elsewhere, where he comes in contact with the infection already developed in another dog, and brings it back in his system.

The development anew of infectious qualities in previously saprophytic bacteria may be favored by inhalation of irritant, gritty road-dust ; of smoke or irritant gases ; or it may result from laceration and concentrated, septic fermentation in foul ulcers of the gums, mouth or throat. It is further claimed that the sudden appression of an eczema on the skin, may be followed by a bronchitic eruption which becomes the occasion of the microbial invasion.

Symptoms. These accord with the portion of the air passages attacked and the gravity of the affection. There may be first a simple rhinitis with sneezing and foul muco-purulent discharge from the nose ; or it may be an ulcerating mouth or tonsils with extreme fœtor and even retching or vomiting ; this is followed by cough, with or without sore throat aggravated by pressure on the larynx or trachea. This cough is at first small, dry and painful, becoming later of a mucous or gurgling character with a profuse discharge. Mucous and blowing sounds are heard in the lungs without any material change of resonance on percussion. From this point it follows the regular course of simple tracheo-bronchitis.

Symptoms in capillary bronchitis. The gravity of the symptoms increases as the disease extends to the smaller bronchia, and the dog is specially liable to such extension by reason of the compact structure of the lung, the comparative absence of loose connective tissue, and the rapid subdivision of the bronchia to their smallest ramifications. For the same reason extensions to the lungs take place readily. When the capillary bronchia are invaded, the breathing becomes more rapid and sighing or panting, the patient sits on his haunches with fore legs apart, and elbows turned out, or he stands obstinately like the horse. In any case he avoids the normally self-indulgent attitude of the healthy dog, that of extension on his side.

The cough is very frequent and paroxysmal, and contributes to frothing around the lips, tinged or streaked with blood. The abundant and even bloody muco-purulent nasal discharge often dries, during the night, around the nares and for some distance inward, obstructing the passage of air, and causing breathing through the mouth, often with marked inflation of the cheeks and flapping of the lips. Retching and vomiting are not uncommon. Percussion may detect no change though circumscribed areas of flatness may betray the existence of collapse through blocking of a bronchus. Auscultation gives especially mucous râles coarse and fine, wheezing, sibilant or cooing sounds, strident ronchi, and even at times a suggestion of a coarse crepitation. The temperature may reach 105° F. but is irregularly variable, the prostration of the dog is great, the eyes lusterless and sunken, the face drawn and anxious, and the animal dull, stupid and even partially comatose, and unable or unwilling to stand on its feet. He usually lies upon his breast with head and fore limbs extended to favor breathing as far as possible. Emaciation goes on rapidly, and in bad cases the dyspnoea goes on increasing to a fatal issue.

Diagnosis. The preservation of life, spirit and appetite, of a healthy resonance over the chest on percussion, of a normal respiratory murmur, in the presence of the mucous râle in the larger bronchia, and a free muco-purulent discharge bespeaks a tracheo-bronchitis, or at least a bronchitis of the larger bronchia only. The presence in addition of dyspnoea, cyanotic mucosæ, areas of flatness on percussion from collapsed lung, fine mucous râles, sibilant, or cooing sounds, ronchi, painful cough, great prostration and dullness, rapid emaciation, flapping lips and cheeks, and threatening asphyxia bespeaks capillary bronchitis.

Prognosis. Inflammation confined to the larger bronchia is usually mild and may terminate in recovery in a week or ten days. Capillary bronchitis on the contrary is liable to cut off the patient early by asphyxia, or to be prolonged with great attendant debility, auto-poisoning, low fever, and a fatal extension to the lung tissue, and bronchial and mediastinal glands. A fatal issue is very frequent in this form.

Lesions. In *tracheo-bronchitis* the lesions often do not extend beyond congestions and arborization of the mucosa, and the ac-

cumulation of muco-purulent matter in the upper air passages. Some congestion and swelling of the bronchial glands may also be noted.

In *capillary bronchitis* the larger capillary tubes, of a millimetre and over, show a tumid, congested mucosa and submucosa, with a considerable collection of muco-purulent matter. In the smaller bronchia, less than a millimetre in diameter, there is a similar congestion and swelling but the tubes are usually filled, and even abnormally distended with the muco-purulent product containing an excess of small, round, granular exudation corpuscles, which also fill the submucosa. In the minute terminal tubes opening into the air sacs and air cells, the air passages are filled with the same inflammatory exudate which extends into the alveoli as well, constituting dirty white or yellowish centres in the midst of a general red consolidated lung tissue into which the inflammation has extended. In short, the case has merged into broncho-pneumonia. Other lesions in such lungs are collapse, from the blocking of individual bronchia by inspissated mucus, emphysema, lobular and interlobular, determined largely by the spasmodic coughing and the frequent sudden over-distensions of the already paretic air sacs and alveoli. It may also be charged in part on the hurried and forcible inspirations, which are the efforts to introduce a sufficiency of air into lungs which are in many parts, obstructed, blocked and collapsed, so that the still pervious portions are necessarily over-distended under the violent efforts. Combined with the other elements of the abundant exudate into and around the capillary bronchia and alveoli, are numerous microorganisms which may vary in different cases and outbreaks and represent the progeny of the original saprophytes which have become pathogenic under the existing environment.

Prevention. To obviate outbreaks of infectious bronchitis, precautions must be taken against infection from outside in shows and other public gatherings of dogs, in travelling by rail or boat, in sending into other kennels or packs for service or other purposes, or in being placed in public buildings in which infected dogs may have been kept. When the animal which has been thus sent from home is returned to its home and companions he should be first placed alone in quarantine for two weeks, and bathed with an antiseptic lotion before he can be considered safe.

In the same way all dogs, and especially such as have been exposed, should be guarded against those accessory causes which most commonly open the way for the entrance of infection. Over fatigue, ice-cold baths, cold draughts when heated and exhausted, damp, underground cellar beds, cold stone or iron sleeping place, decomposing or insufficient food, ulcerative diseases of the mouth or throat, starvation, low condition, debility from previous ill-health, and irritation of the air passages by irritant dust, or gases, overheated air, smoke, etc., must be as far as possible avoided.

Treatment. The curative treatment must be that in common use for simple bronchitis, with special attention to the use of antiseptic inhalations or injections. The patient must be protected against cold, outdoor, winter, or damp air, and especially rains and snows; currents of air must be avoided, and even customary baths must be for the time abandoned. Yet the kennel must be pure, sweet, and well-aired, and if this can only be secured at the expense of a lower temperature a coat may be put on the patient.

As a soothing agent use water-vapor from a jet of steam thrown into the chamber, and impregnation with an antiseptic (sulphur fumes largely diluted, carbolic acid, creosote, terpene, terpinol, cresyl, oil of turpentine, eucalyptol, chlorine, iodine) will greatly enhance its value. Leaves of elder in decoction have been used to render the water-vapor more soothing and diaphoretic, and anise, poppy heads and even opium have been used to serve a similar soothing purpose.

Or direct expectorants may be given by the mouth, ipecacuan wine, 10 to 20 drops, syrup of senega, a teaspoonful, or apomorphia, $\frac{1}{100}$ grain. Or ammonium chloride may be vaporized by heat and inhaled by the patient. A hot, damp pack of the chest with damp cotton covered with a dry dense material and maintained in close contact with the skin by elastic bandages will at once soothe the bronchia and solicit expectoration. It can only be safely used in a warm room and when taken off must be done one part at a time, and the coat thoroughly dried and covered with a warm dry wrapping. As an expectorant Cadeac recommends essence of anise 1 gram, aqua ammonia 5 grams, alcohol 24 grams. A mustard poultice on the sides of the chest will often serve an excellent purpose, or they may be rubbed with common soap

liniment. When the discharge has been freely established, opium ($\frac{1}{8}$ grain) or muriate of morphia ($\frac{1}{10}$ grain) may be given, or it may be added to the agent inhaled. Cadeac combines the morphia with prussic acid as follows: Morphine 25 centigrams, water of bitter almonds 10 grams, water 150 grams. Dose, two to three tablespoonfuls in soup or tea daily.

It must not be forgotten that the system is being charged with toxins and the products of rapid tissue change, and these must be eliminated to prevent dangerous or fatal auto-poisoning. This may be most safely secured by an abundant ingestion of water. To this end a warm milk diet or soups will contribute. Cadeac recommends a free use of an infusion of tea or coffee. If these are refused, direct diuretics may be given such as nitrate of potash 5 grains, or even iodide of potassium in similar amount. If the patient is weak or debilitated, sweet spirits of nitre, or liquor of acetate of ammonia may be substituted.

In case of very high fever which is not lowered by the antiseptics, and elimination, a few five grain doses of acetanilid may be given. In weak conditions of the heart, on the other hand, strychnia, $\frac{1}{100}$ grain, or digitalis, $\frac{1}{2}$ grain, may be given twice a day.

In the second stages of the disease and during convalescence muriate of ammonia, five grains, with liquorice, five grains, will prove of great value.

For general infection Fortuna advises: essence of eucalyptus, twenty grams, sterilized olive oil, eighty grams. Dose: one to two cubic centimetres according to size, subcutem daily, or exceptionally twice or thrice. Simultaneously he advises: Naphthol B., ten grams, bismuth salicylate, six grams, pulverized sugar, twenty grams. Mix and divide in fifteen. Give three to six daily.

Finally, as in simple catarrhal bronchitis, in the advanced stages, with much prostration and debility, stimulants (sweet spirits of nitre, tincture of gentian, port or sherry wine), and tonics (syrup or tincture of iron, liquor sodæ arsenitis, echinacea, or cod-liver oil) may be used.

CHRONIC BRONCHITIS IN THE DOG.

Causes. Chronic bronchitis is usually seen in old age in fat, pampered dogs, obese, and subject at the same time to chronic eczema. It has been supposed therefore that the morbid products absorbed from the diseased skin produce irritation of the bronchial mucosa while being eliminated by that channel. Again the intimate nervous sympathy between the skin and its internal prolongation—the mucosa—has been adduced as the cause of the coincidence, just as disorders of the gastro-intestinal mucosa usually lead to cutaneous eruptions. We must not, however, forget a third condition, namely, the plethoric condition of the pampered pet, the common disorder of liver, stomach, intestines and kidneys, and the loading of the blood with waste products of metabolic changes, which should have been eliminated, and the morbid products of disordered digestion, hepatization, and sanguification, which act injuriously on the skin and the bronchial mucosa alike.

As in other animals obstructions to the return of venous blood from the lungs, determine chronic congestions of the bronchial mucosa, hence valvular diseases or insufficiency, of the heart ; pericarditis ; lymphadenoma, tubercle, or other swelling of the bronchial glands become efficient causes.

Symptoms. The symptoms vary according to the dryness, or secretion from the mucosa. In the first form the cough is frequent and paroxysmal, but husky and dry. In the second it is mucous, soft, gurgling accompanied by more or less muco-purulent expectoration and not infrequently with vomiting. The discharge is sometimes fœtid and may block the nasal passages, or agglutinate the margins of the nostrils.

The physical symptoms are like those met with in acute bronchitis. Fever is usually absent though a slight rise of temperature may be present at intervals.

The disease may last for years, becoming aggravated in winter and improving with genial summer weather. In the fat, pampered old dog, the disease is usually replaced in a measure by the cutaneous eruption, while in winter the eczema moderates and bronchitis comes to the front.

Course. Prognosis. The disease being so frequently the consequence of age, idleness and obesity, it is of an obstinate character, and too often proves altogether incurable.

Lesions. These are the common attendants on bronchitis, except in the matter of congestion and redness. There is usually emphysema, the affected lobules projecting beyond the average level, and of a very pale color; collapse of certain lobules marked by flattening and flesh-like color; dilatations of the bronchia with accumulations of more or less tenacious exudate; constriction of the bronchia from peribronchial exudation and hyperplasia, and, it may be, thickening of the bronchial mucosa itself. The mucosa is uneven in outline, of a dull gray color, and it may be covered with minute papillary hyperplasia. As in acute bronchitis, bacteria are invariably found in the diseased tissues and discharges, and contribute largely to the obstinacy of the case and the difficulty of treatment.

Treatment. For successful treatment the first consideration is the removal of those causes that lay the system open to the affection and serve to maintain it. If the patient can be put on a simple and restricted diet and, in a genial season, can have abundant out-door exercise, the obesity may be reduced and digestive and hepatic functions improved. Even when a patient is not at first able to take active exercise, he may be taken out in a carriage until he gains vigor enough to run by himself. In the same way regular warm baths (70° to 80°) and liberal massage daily may greatly reduce corpulency and improve the health. In an acute access, however, exposure in cold weather is to be carefully avoided and a moderate even temperature secured. In such cases massage and mild saline laxatives (Rochelle salt) daily will serve a good purpose. To dissolve and loosen the expectoration we may use expectorants (ipecacuan, Dover's powder, apomorphia, senega, liquorice, horehound), or the more stimulating expectorants (turpentine, terebene, glycoheroin, balsam of Peru or Tolu, or anise).

Inhalations of tar, turpentine, carbolic acid, creolin, cresyl, lysol, eucalyptol may be of great use.

When the cough is very annoying, calmatives and sedatives are called for (opium, codeine, morphine, Dover's powder, dilute

hydrocyanic acid, bromides, hyoscyamus, conium, belladonna, stramonium). A combination of the soothing and expectorant agent may be used : Apomorphia, 1 gr.; muriate of morphia, 6 grs.; dilute hydrocyanic acid, 30 drops; distilled water, 2½ ozs. Dose, one-half to one teaspoonful every three hours. Or sulphate of morphine, 2 grs.; syrup of squills, 2 ozs.; syrup of wild cherry bark, 2 ozs. Dose, teaspoonful every three hours. Or without the sedative; Muriate of ammonia, 2 drs.; extract of liquorice, 2 drs.; water, 4 ozs. Dose, a teaspoonful every three hours. Again a stimulating expectorant may be adopted; Carbonate of ammonia, 1½ drs.; sulphate of morphia, 1 gr.; spirit of anise, ½ oz.; syrup of wild cherry bark, 3 ozs. Dose, teaspoonful every three hours.

In extreme conditions oxygen inhalations are of great value.

Much benefit can often be derived from a long course of arsenate of soda, strychnia or cod-liver oil.

CROUPOUS BRONCHITIS IN CATTLE AND SHEEP.

Causes : smoke, hot air or gas, irritant inhalations, concomitant of infectious diseases. Lesions : intense congestion covered by fibrinous exudates. Symptoms : slowly or suddenly developed, fever, loud, wheezing, stertorous, panting breathing, dyspnoea, dry râles and blowing. Course. Treatment : moist jacket, soothing, expectorant, stimulant inhalations, expectorants, derivatives.

This affection has been found in cattle and sheep from exposure to smoke, hot air or gas, and other irritants, and even from exposure to cold, and without any suspicion of a contagious element. Again it has been seen as a complication in Rinderpest, lung plague and malignant catarrh. Gamaleia found it in sheep associated with *diplococcus lanceolatus* and Mayrwieser as an epizootic in all the animals of a brewery. I have seen it in a valley in Central New York on watersheds running north to Lake Ontario and South to the Susquehanna, at midsummer, implying an enzootic cause, yet not attacking all the cattle, but only a few in a herd. In these cases the malady was very fatal, the bronchi and bronchia becoming completely filled with false membrane so that the animal died asphyxiated. The lesions are those of tumefaction and extreme arborescent redness of the mucosa, and the formation of patches of a dense fibrinous exudate of a yellowish color, in some cases completely obstructing the tubes. The false membranes are friable and putrefy quickly. Besides a fibrinous basis, they contain leucocytes and red globules more or less altered, and an abundance of micrococci. Cadeac inoculated the latter on the Guinea-pig on two occasions without effect. The bronchia in which there were no false membranes contained a tenacious mucus streaked with blood.

Symptoms. The attack may come on slowly as in ordinary bronchitis, while in other cases it is sudden. The respiration is loud, wheezing, stertorous and panting, general dyspnoea often supervening. Auscultation furnishes loud, blowing sounds, dry râles and ronchi, while percussion may show no abnormal change. A strong tremor is felt by the hand on the trachea, and after a paroxysm of coughing false membranes may be expelled. If

there is no improvement by the second or third day death is liable to supervene by asphyxia.

Course. The progress is very rapid and almost always to a fatal termination in from one to three days, especially in the young. In case the mass of false membranes is coughed up a recovery may be hoped for. The absence of additional croupous product in the bronchia is indicated by the subsidence of the extreme dyspnœa and the improvement of the general symptoms.

Treatment is usually unsatisfactory. The hot, moist jacket, inhalation of vapors or warm water, of carbonate of ammonia, and of ether may be tried, counter-irritants to the chest, and internally, liquor ammonia acetatis and iodide of sodium would be indicated. When the membranes are somewhat loosened pilocarpin, or in weaker subjects apomorphine, may assist their expulsion. Peroxide of hydrogen may be injected into the trachea with due caution against sudden asphyxia.

ACUTE CONGESTION OF THE LUNGS. PULMONARY HYPERÆMIA.

Active and passive congestions. Congestion of incipient pneumonia. Congestion of over-exertion. Causes: lack of condition, fat, plethora, gorged stomach, hot weather, cold rains, cold baths, infectious diseases. Symptoms: dilated nostrils, labored breathing deep lifting of flanks, panting, pendent head, staring, fixed, bloodshot eyes, pale—later dark red nasal mucosa, rapid pulse, palpitating heart, fine crepitation, cold limbs, tremors, perspiration, obstinate standing till unable, blood from nose. Fulminant cases. Exposure cases. Course. Termination. Resolution. Lesions: lungs black, gorged, do not crepitate nor collapse, lessened buoyancy, cut surface, compressed bronchioles and alveoli, right heart and veins gorged, blood black, semi-liquid, petechiæ. Nature. Not yet inflammatory, blood engorgement, no cell proliferation, migration, non-exudation, pulmonary vaso-motor paresis, effect of blood pressure, of peptones, etc., of exhaustion. Treatment: relieve respiratory muscles, derivation to surface, stimulants, hot pediluvia and packs, relief of vascular system, bleeding, heart stimulants, digitalis.

Congestion of the lungs occurs in all animals as the precursor of inflammation, but as death may occur without the supervention of actual inflammation a special notice appears to be demanded. The hyperæmia of the lungs may be seen in two forms, active and passive, the latter form being secondary to other diseases, such as valvular diseases of the left heart, by reason of which the blood is forced back on the lungs and creates mechanical congestion. The active form is a pathological process developed in the lung itself, and which often proves fatal through arrest of the circulation through this organ.

Causes. The pulmonary congestions preceding pneumonia are due to the same causes with that disease. The most typical, acute and deadly form of pulmonary congestion is usually due to *over-exertion in an animal that is fat and out of condition.* The English hunting field presents the most typical specimens. A horse that has just left the dealer's hands, or that is plethoric, fat, soft and flabby, is ridden over a heavy country, and though he may perform well for a few miles, he soon hangs heavily on the bit, slackens his pace, and if not pulled up, staggers and falls "all of a heap." A farm horse, taken from grass or other soft

feeding, and entirely out of condition often suffers in the same way, in going perhaps for the veterinarian in case of urgent colic in one of its fellows. Cruzel draws attention to similar congestions from over-exertion in fat cattle, and Trasbot in wild stags and hogs when beechnuts and acorns were abundant, in pampered family horses and in plethoric farm animals generally. Excessive heat (heat apoplexy) is invoked as a cause, and the arrest of hæmatisis and consequent stagnation in the pulmonary capillaries are undoubtedly accessory causes, yet the majority of cases, and the most typical and fatal, occur in the winter season (the hunting season). On the other hand, chills from rain or cold draughts, especially when heated and exhausted, are common causes, and the disease often comes on more gradually, attaining its acme after five or six hours. A horse perspiring after a hard drive and left to face a cold blast unblanketed, or one plunged by accident into ice cold water for ten minutes (Trasbot) are examples of this kind. These cases are ushered in by violent rigors, whereas in those due to over-exertion this is much less marked and is usually only suggested by the coldness of surface and extremities. Another condition which contributes to pulmonary congestion is a full stomach. The plenitude of the abdominal organs leads to compression of the lungs and hampered circulation, and when to this is added over-exertion and exhaustion acute congestion is speedily induced.

Acute congestions are noticed as an accompaniment of other diseases, but these are mostly either the localization in the lung of a specific morbid process (anthrax, influenza, distemper, strangles), or it is due to auto-poisoning, as when the cutaneous transpiration is suppressed by a coating of glue, or to embolism.

Symptoms. In the horse which fails under severe exertion there are the dilated nostrils, the labored breathing, the deep, almost convulsive action of the flanks, the hanging on the reins, the slackening of the pace, the unsteadiness of gait, and lastly the fall. There may now be noticed the protruded bloodshot eyes, the agonized expression of countenance, the extended head, the pallor, and later the blueness of the nasal mucous membrane, the short, panting breathing, accompanied by a roaring noise alike in inspiration and expiration, and the small, weak, rapid pulse often imperceptible at the jaw. If the animal has been stopped short

of having fallen, or if he is able to get upon his feet, he stands with his limbs apart to secure his stability, and with the elbows turned out to facilitate the expansion of the chest. As the breathing becomes panting the respirations are less deep, the ribs are maintained permanently drawn outward, and the flanks rise and fall to a limited extent only, but with great rapidity (eighty to one hundred per minute). Auscultation may detect at first an increase in the pitch of the respiratory murmur, and the presence of the finest possible *crepitation sound*. Soon the murmur decreases uniformly. The extremities are cold, and in this coldness the general surface to some extent participates even though it may be covered by perspiration. Tremors or rigors are present. The heart is felt behind the left elbow to beat tumultuously. If blood is drawn it flows in a thin, black, tarry stream.

In some cases blood more or less frothy is discharged from the nostrils as the result of rupture of pulmonary vessels.

In the fulminant cases in **cattle** respiration is rapid, even panting, wheezing, the expiration attended by a hoarse grunt, sometimes nasal hemorrhage, great prostration, profuse perspiration, a stupor sets in and the animal falls and dies, with more or less struggling.

In the cases which develop more slowly, and as the result of cold and chill whether in horses or cattle, there is dullness, anorexia, prostration, increasing rapidity and oppression of the breathing, a small, frequent, hoarse cough, and at first distinct pallor of the conjunctiva and pituitary mucosa, with more or less trembling. The head is extended on the neck, toward an open door or window, if available, until prostration and stupor forbid. The pulse is small, thready, often almost imperceptible and much accelerated, while the heart beats are strong, violent, tumultuous. For a time the respiration may not be more than double the normal rhythm, but it tends to more or less rapid increase with wheezing or stertorous sounds and shaken by trembling of the respiratory muscles. The nasal discharge is slight and grayish often with streaks of blood. If it increases the cough becomes looser and softer. Quite early the respiratory murmur decreases over the whole lung and a blowing sound from the bronchia or larynx is heard on the upper middle third of the chest. This may be complicated by

a mucous râle, or when hæmorrhage has supervened by a loud rattling. Percussion shows a lack of resonance over the whole lung, not so flat and definitely circumscribed as in pneumonia but a partial flatness of sound over the whole chest. In pneumonia a limited area of lung is absolutely solidified (hepatized) while the remaining lung is practically normal, whereas in acute congestion often the whole lung is gorged with blood, but for some time no part of it is entirely divested of air.

Another marked feature is the maintenance at first of the normal temperature with only a slight rise of about 1° . This serves to distinguish congestion of the lungs from sunstroke (heat anhmæmatisis) in which the temperature usually rises to 108° 110° F. or higher. The temperature rises however as the disease advances and merges into pneumonia. Another distinguishing feature from sunstroke is the early pallor of the mucous membranes which in heat apoplexy are strongly congested. In congestion they become dark red only with the advance of the disease and the advent of asphyxia. These features serve also to distinguish acute pulmonary congestion from contagious fevers, pneumonia and other inflammations of internal organs.

Course. Termination. The more acute (fulminant) forms are promptly fatal. In the exhausted system the lungs have become uniformly gorged with blood, which can no longer be forced through the capillaries by the right heart, the heart in turn is over-distended with blood and ceases to beat and death ensues in a few minutes.

In the less acute cases the patient survives twenty-four hours and upward, the whole lung not being equally implicated but only certain lobules, usually the lower, or the congestion, if uniform in all the lung, being less extreme.

In favorable cases recovery takes place in one or two days. There is a return of life and appetite, a gradual improvement in pulse and breathing, the respirations becoming deeper and longer, and in a few hours all the more violent symptoms may have disappeared. With a more gradual improvement recovery may still be complete in four or five days.

Lesions. When the subject has died suddenly, the appearances are essentially those of uniform engorgement of the pulmonary capillaries with blood. The general aspect is a dark red, varying

from reddish brown to black, the darkest shades corresponding to circumscribed areas of actual hæmorrhage. In the worst cases the whole mass may appear like black current jelly. The lungs do not collapse when the chest is opened, they are more or less friable at various points, and different portions will sink or float in (not on) water, according as it may be more or less airless. A dark liquid blood exudes freely from the torn or cut surface. Sections of the lung tissue hardened and examined under the microscope show the alveoli and bronchioles devoid of exudate, but having their cavities compressed and obliterated by the pressure of the swollen mucosa, and its investing blood clot. The heart is over-distended with fluid blood. In asphyxiated cases the general venous system is filled with black, liquid blood, and the serosæ spotted with petechiæ.

Nature. The nature of this disease is variously understood. It differs from inflammation in the absence of active cell proliferation and migrations, of inflammatory exudation, and of fever at all proportionate to the extent of the lesions. All these may and do supervene if the patient survives but they are practically absent for a length of time at the outset. Some attribute it to paresis of the vaso-motor centres for the lungs, as the result of their over-stimulation and of the retrocession of blood from the chilled surface to the internal organs. But congestions caused by cutting the cervical branch of the sympathetic nerve or the sciatic plexus are not marked by a similar blood extravasation and destruction of tissue. The delicate structure of the lung tissue and the comparative absence of mechanical support will account for this in part, the great force of the circulation overloading the capillaries, under the impulse of the heart so closely adjacent, has doubtless a certain effect, and the venous nature of the blood thus forced into the lungs and calculated to arrest all normal function has a potent influence. If we add to this in the *over-exertion* cases, the sudden advent into the circulatory stream of unchanged peptones and other ingredients of the portal blood of highly fed and plethoric animals we find a sufficiently pathogenic combination. In all acute cases, however, the adiposity, poor condition and susceptibility to speedy exhaustion must be given their full share of responsibility.

Treatment. Girths, saddles and anything else that may hamper

the movement of the chest must be at once removed and the horse's head turned to the wind, an active stimulant given and the legs well rubbed and loosely flannel bandaged. The nature of the stimulant is of less consequence than its prompt administration. Two ounces of chloroform, of sulphuric ether, or of sweet spirits of nitre; half a pint of whisky, brandy or gin; or a pint of any of the more stimulating wines may be given, diluted in warm water so as to remove their irritating qualities. A drink of warm gruel will often go far to restore warmth to the surface and to unload the overtaxed lungs. Frequent large injections of warm water have a similar effect. Active hand rubbing of the legs and the wrapping of them loosely in flannel bandages previously warmed at the fire are equally valuable. If a roomy, well ventilated, loose box can be obtained the horse should be led to it gently and a light but warm rug placed upon the body. Valuable derivation may be obtained from pediluvia, the feet and legs up to the knees and hocks being put in buckets of water as hot as the hands can bear, and at the same time actively rubbed. If this is impossible the legs may be wrapped in bandages and wet with hot water every few minutes. Or this soothing derivative agent may be applied as well to the surface of the chest. A blanket wrung out of hot (nearly boiling) water until it no longer drops is wrapped round the body and covered up with two or three dry rugs. A second smaller rug is wrung and placed on the neck and covered by a sufficiency of hoods to keep in the heat. The legs are meanwhile hand rubbed and bandaged and the other measures above recommended carried out to restore the circulation in the surface and extremities. The time-honored practice of bleeding freely from the jugular vein is one of the most effective means of relieving the over-charged heart and lungs, and should be resorted to at the earliest possible moment. The blood will at first flow in a small, dark stream, but as the circulation obtains relief the jet will increase in volume and the general symptoms will improve. From four to six quarts may be taken with advantage from an ordinary horse. This is not a pneumonia but an overloaded condition of heart and lungs, threatening speedy death and which the abstraction of blood promptly relieves.

The longer the bleeding is delayed the less effective it is. It should not supersede the other measures already recommended.

There is no real paradox in both bleeding and giving stimulants in such a case, as the essential condition is one of weakness, and if the abstraction of blood has been of use in relieving the clogged heart and lungs, the depression under which these have labored may be still further overcome by agents calculated to rouse their suspended vitality.

Trasbot strongly recommends large doses of tartar emetic and iodide of potassium to reduce the blood pressure in the lungs, an advice which will be received with hesitation by those who dread the already paretic condition of the heart. His combination of iodide of potassium with digitalis will be more confidently resorted to. One drachm of the former may be given with a half drachm of the latter twice daily.

With the advent of marked fever and other signs of pneumonia, the treatment for that disease should be resorted to.

PULMONARY ŒDEMA.

Causes : pneumonia, extra force of right heart or weakness of left, insufficiency of mitral valves, deflection of blood by obstruction in one portion of lung, pressure of tumor on pulmonary veins. Malignant œdema. Malignant catarrh. Bright's disease. Anæmia. Parasitism. Symptoms : abnormal heart sound, or urinary secretion. Parasitism. Percussion shows flat sound, auscultation lowered respiratory murmur, heightened blowing. No crepitation. Expectoration serous. Prognosis grave. Treatment : attack primary disease. Posture. Elimination. Dry cupping. Heart tonic.

A dropsy of the lung tissue may supervene in weak conditions, in the course of inflammatory disease of the lungs ; it may also depend on an imperfect balance in the forces of the right and left heart respectively, which leads to the habitual throwing of blood pressure back upon the lungs. Still more frequently the congestion and dropsy depend on insufficiency of the mitral valves by reason of which a reflux of blood toward the lungs takes place at each heart-beat. The pressure of tumors on the pulmonary veins may have a similar action. Obstruction of circulation in one portion of lung may cause an extra blood pressure on an adjacent one, and œdema so caused may be found especially in cattle and pigs, in which the interlobular connective tissue is specially abundant. This may be seen in miliary tuberculosis in cattle, and it probably contributes to produce the extraordinary liquid collections that characterize lung plague. In cattle also malignant œdema or hæmorrhagic septicæmia may affect the lung, and an œdematous condition is sometimes met with in malignant catarrh. Bright's disease is another cause, the uræmic dropsy finding a favorite seat of election in the loose lung tissue unsupported by solid structures. The anæmia resulting from parasitism, like distomatosis, may similarly affect the lung.

The *symptoms* of pulmonary œdema will usually be complicated by those of the affection causing it. Thus modification of the first heart sound or of the urinary secretion, or the existence of parasitism, would furnish valuable indications.

The *physical signs* of lung œdema vary. If pneumonia is present it is betrayed by its characteristic symptoms. In the absence of inflammation there is dullness on percussion over the

affected area, and on auscultation an absence of the respiratory murmur, and perhaps abnormal clearness of bronchial, cardiac and other sounds from deeper parts. It differs from pneumonia in the absence of fever and of any crepitation surrounding the consolidated portion. The expectoration is serous or watery, rather than rusty or purulent.

The *prognosis* is always grave in proportion to the incurable nature of the primary disease. Chronic valvular or Bright's disease, miliary tuberculosis or malignant tumors would render the case hopeless, while in acute pneumonia, or nephritis or parasitism there may be some hope. The *treatment* will largely consist in the therapeutics of the primary disease, yet we may also seek to relieve the dangerous symptoms of œdema. The frequent change of position may serve to limit hypostatic accumulation. Diuretics or purgatives in strong patients will favor absorption. Pilocarpin more than any other agent secures temporary absorption, but cannot be continued owing to its depressing effects. Digitalis is often valuable in improving the heart's action and acting freely on the kidneys. Dry cupping on the chest acts as a derivative.

ATELECTASIS. COLLAPSE OF LUNG.

Atelectasis in bronchitis, congenital, etc. Airless condition in the absence of exudation. Causes : congenital persistence in butcher animals. Blocking of air tubes by exudate—ball valve. Desquamation of ciliated epithelium. Compression by hydrothorax, pneumothorax, and false membrane. Symptoms : percussion flatness, juvenile respiration elsewhere, blowing sounds loud : drum-like sounds on emphysema and pneumothorax, cyanosis. Lesions : depressed, flesh-like, non crepitating lobules or lobulets, sink in water, dilatable. Treatment : rouse respiratory centres, douches, cold and warm, slapping, electricity, forced inspiration, diet, massage. Treat attendant disease.

This has been already referred to as a result of bronchitis, but it deserves special mention as a sequel of that affection, and in various domestic animals, as an independent condition. The condition is one of consolidation of lung by the complete exclusion of air, but without any infiltration of its substance by inflammatory exudate or dropsical effusion. The tissue remains in its normal state apart from the fact that its bronchioles and air sacs are undilated. The affected portion has a solid dark fleshy appearance. The collapsed portion often represents one lobule or group of lobules which communicate with a single bronchium.

Causes. In some instances the conditions remain from birth, the lobule never having been called into use. This is seen especially in cattle and other meat producing animals, in which active breathing is systematically suppressed in the interests of rapid growth and the deposition of fat. In the improved breeds the lungs remain larger than the exigencies of the life demand, and large portions remain out of use. In bronchitis the condition is acquired, and is mainly dependent on the blocking of a bronchial tube with tenacious or semisolid mucus. The pathological lesions of bronchitis favor this since one of the earliest changes in the inflamed mucosa is the desquamation of the columnar epithelium. This removal of much of the cilia, and the paralysis of much of what is left, annihilates for a time the normal method of clearing away the secretion, and this being now produced in excess, blocks the tubes. This secretion virtually acts like a ball valve in favoring the exit of the air during the convulsive expiration of coughing, and hindering its entrance during the

succeeding inspiration. The bronchia and bronchioles decrease in size to near their termination, so that, as forced out in coughing, the secretion enters the larger tube and allows the exit of air, while as drawn back in inspiration it enters the smaller tube and closes it against any possible ærial entrance. Mendelssohn and Traube demonstrated this action by introducing shot into a dog's lung, and in two days the left lung was found collapsed and the right one the seat of complementary emphysema. The violence and frequency of the cough therefore, bears a ratio to the occurrence and extent of atelectasis. Other causes are the compression of the lower lobes of the lung by hydrothorax, by pneumothorax (developed by lacerated lung or perforated chest wall), or by a false membrane covering the lobe and contracting in process of organization.

Symptoms. As occurring congenitally in the improved meat producing animals the condition is rarely recognized in life and cannot be said to be a defect. The collapsed lobule being farther removed from the air may be a more favorable field for the growth of pathogenic bacteria, but on the other hand these do not so readily penetrate it as if the tubes were open. When the collapse is more extensive, the contrast in the flatness on percussion and indistinctness of the respiratory murmur on the affected side, and the marked resonance and loud murmur on the other, may serve to identify the affection. In extensive, traumatic cases this contrast is much more prominently marked, as the expanded portions have to take on extra compensatory work and are not infrequently rendered emphysematous. The drum-like sound in percussion of such parts, and in the upper part of the chest in pneumothorax is pathognomonic of these conditions. Again in hydrothorax the horizontal upper level of the area of dullness betrays a liquid cause. Severe cases are marked by cyanosis.

The *lesions* seen in atelectasis consist of depressed areas of a dark fleshy color on the surface of the lung, usually sharply limited by the borders of the lobules, and in strong contrast with the bulging, light colored lobules adjacent, which are often emphysematous. The collapsed lobule may usually be dilated when air is forced into the bronchium, but if it has been of some standing this is often difficult or impossible. If it has resulted from bronchitis or compression of a previously inflated lung it will often

float in (not on) water, from a little retained air, but in congenital atelectasis it is airless and sinks to the bottom.

When *treatment* is demanded it will vary according to the cause. In congenital atelectasis the respiratory centres must be roused. The new born animal may be sprinkled alternately with ice-cold and hot water, or the chest may be slapped with the palm of the hand or a wet towel. The nostrils must be cleared of mucus, and the lungs inflated by blowing with bellows, the larynx being pressed back against the gullet to prevent inflation of the stomach. If available, electricity may be applied to the chest walls. These measures may be repeated at intervals and the systemic weakness overcome by nourishing food, stimulants and friction of the skin.

In acquired atelectasis we should seek to correct the disease to which it owes its existence. In bronchitis the measures already indicated for the liquefaction and removal of the expectoration will be in order; in hydrothorax a judicious paracentesis and in pneumothorax the aspiration of the gas, and the closure of any traumatic opening through which that gas has gained access.

HÆMOPTYSIS.

Causes: over-exertion in plethoric, glanders, pulmonary tubercle, petechial fever, embolism, aneurism, ulcerated new formations, anthrax, septicæmia, hæmorrhagic diathesis. Symptoms: inappetence, cough, cold limbs, rigor, hard pulse, jugular pulse, violent heart beats, unsteady gait. Discharge, bloody, crimson, frothy, with cough, without acid, excited breathing, debility. Indications from pre-existing disease. Treatment: quiet, elevated head, cold irrigation, ice bags, acetate of lead, lime salts, opium, ergot, matico, tannin, iron, oil of turpentine, laxatives, cool stable.

The term *hæmoptysis* (*αιμα*, blood, *πτυνω*, I spit,) is now entirely restricted to bleeding from the lungs and lower air-passages. It is a very rare complaint in the lower animals, but is sometimes seen in both horse and ox. In very plethoric subjects the overloaded circulatory organs give way in the delicate membrane, lining the ultimate bronchial tubes and the air cells. The exciting cause in such cases is usually some severe effort of draught, a violent gallop, or other unwonted exertion. It occurs in glanders from rupture of caseated pulmonary nodules. It does not appear to be so common in phthisis in the lower animals as in man, but one case occurred, under the eye of the writer, in which the bursting of a large tubercle in the lung of a cow involved the rupture of a considerable vessel with a fatal result. Pulmonary embolism and infarction, petechial fever, aneurism, ulcerated neoplasms, anthrax, and septicæmia are additional causes. Lastly hæmoptysis sometimes takes place in hæmorrhagic subjects without any appreciable rupture of vessels, the blood sweating from the surface of the bronchial mucous membrane.

Premonitory symptoms are sometimes noticed, such as dullness and lassitude, loss of appetite, a frequent short cough, coldness of the limbs and surface, shivering, full, hard pulse, pulsation in the jugulars, tumultuous action of the heart and unsteadiness of gait.

More commonly it comes on suddenly as the result of severe muscular strain or excitement. The blood flows from the nose and rarely from the mouth in solipeds, but indiscriminately from both in other animals. It is bright red, clear, frothy, or mixed with mucus, and variable in amount. It is easily distinguished

from nasal hæmorrhage, which is not frothy, and from bleeding from the stomach, which is clotted and blackened, with an acid odor from the presence of gastric juice. The cough of hæmoptysis contrasts with the sneezing of epistaxis and the retching of hæmatemesis. The rattling cough increases the discharge, as does also a dependent position of the head. Besides the cough there is usually an anxious countenance, accelerated breathing and considerable lifting of the flank. When the loss is excessive there is weakness, giddiness, rolling of the eyes and pallor of the visible mucous membranes.

The previous ill-health of the patient, the presence of tubercle as ascertained by auscultation and percussion, and the hæmorrhagic constitution as shown by occurrence of bleeding from other parts of the system will lessen the chances of a favorable termination. Sometimes, too, the flow is so profuse that the blood cannot be coughed up, and filling the bronchical tubes it destroys life suddenly by suffocation.

Treatment. When brought on by severe exertion absolute quiescence will usually check hæmoptysis. Keeping the head in an elevated position favors its arrest. The application of cold water to the head, neck and thorax, and the giving of iced water, strongly acidulated by vinegar or one of the mineral acids may sometimes be required. In threatening or obstinate cases one drachm of acetate of lead may be given thrice daily to check by its astringent effect on the vessels, and the addition of a drachm of opium is of great value in suppressing the cough. Ergot, adrenalin, lime salts, gelatine solution subcutem or intravenam, tannin, matico and oil of turpentine have each been employed with advantage, and when costiveness exists a saline laxative (one pound sulphate of soda) may be usefully resorted to. The patient should be kept in a cool, airy dwelling, and should rest for fifteen or twenty days after an attack.

PULMONARY APOPLEXY. HÆMORRHAGIC INFARCTION.

Different forms. Embolism with infarction. Embolism from arteritis. Rupture of blood-vessel. Changes in color. Symptoms. Repair.

Hæmorrhage into the lungs may be : 1st. Petechial in infectious diseases. 2d. *Interlobular* as from ruptured vessels. 3d. *Infarction* or apoplexy. *Infarction* results from embolism of a branch of the pulmonary artery, which may in its turn be due to clots formed in a diseased heart or in the systemic veins and carried to the lungs in the blood stream. It may also result from inflammation of the inner coat of the pulmonary artery. A virtual stasis occurs beyond the embolism, and the blood filtering in through the anastomosing capillaries fills and blackens the affected lobule. With rupture of a considerable vessel the blood escapes *en masse* and appears like black current jelly. As it ages it becomes granular and changes to a yellow color, or it may form a necrotic mass enclosed in a cyst as in lung plague. The symptoms, apart from the absence of respiratory murmur and resonance, are not diagnostic. It may take months to undergo liquefaction and absorption. Iodide of potassium, bitters and stimulating diuretics may be given.

PNEUMONITIS ; PNEUMONIA ; INFLAMMATION OF THE LUNGS.

Definition. Inflammation of the spongy tissue of the lungs uncomplicated by that of the bronchia or pleura.

Divisions. This affection has been variously divided according to *seat, nature, and complications* : thus :

Single Pneumonia : Affecting one lung : *right* or *left*.

Double Pneumonia : Affecting both lungs.

Lobar Pneumonia : Affecting one lobe, or by lobes.

Lobular Pneumonia : Affecting by lobules.

Acute Pneumonia. Subacute Pneumonia. Chronic Pneumonia.

Croupous or Fibrinous : With fibrinous exudate.

Catarrhal : With exudate rich in cells and granules.

Hæmorrhagic : With extravasation of blood.

Purulent : Tending to pus : abscess.

Necrotic : Tending to gangrene : sequestra.

Desquamative : With great proliferation of alveolar epithelium.

Interstitial. Interlobular : Affecting mainly the interlobular connective tissue.

Hypostatic : Dependent on gravitation of the blood.

Metastatic : Due to embolism.

Parasitic : Caused by parasites.

Traumatic Pneumonia : Due to wounds or foreign bodies.

Contagious.

Many of these are, however, but localizations of the same affection, and others are manifestly microbial diseases which in the present state of pathology it is not always easy to early distinguish sufficiently for clinical and therapeutic purposes. For the sake of convenience, therefore, pneumonia will here be treated of more generally, as fibrinous pneumonia, catarrhal or broncho-pneumonia, and inhalation pneumonia, and under the headings devoted to etiology, pathology, therapeutics, etc., attention will be given to distinctions. Those pneumonias that are but pulmonary manifestations of other diseases—influenza, glanders, tuberculosis, strangles, contagious pneumo-enteritis, lung plague, septicæmia, pyæmia, swine plague, hog cholera, petechial fever, actinomycosis, and neoplasms will be considered under these respective headings.

ACUTE FIBRINOUS PNEUMONIA. PNEUMONITIS IN THE HORSE.

Definition. Differentiation from acute vascular congestion. Predisposing causes: age, sex, stabling, training, diet, impure air, low health, previous lung disease, plethora, climate, season. Exciting causes: chill, fatigue, leucomaines, sudor, draughts, plunging in or spraying with cold water, clipping, inhalation of irritant smoke, gas, dust, drawing of food, irritating or insoluble drugs into the lungs, neoplasms, parasites, contusions, fractured ribs, punctures, contagion, plurality of germs, bacillus of Friedländer, micrococcus of Talamon and Fränkel, diplococcus pneumoniae equina of Schütz, diplococcus pneumoniae equina of Cadeac. Symptoms: chill, hyperthermia, dullness on percussion and crepitation in the lower part of the lung, reaction, congested mucosæ, accelerated labored breathing, excited circulation, pulse oppressed, cough deep, patient statant, elbows everted, nose protruded, nostrils dilated, approaching door or window, pinched countenance, skin dry, harsh, adherent, partial sweats, loins insensible, nasal discharge rusty, dependent part of lung largely non-resonant, with peripheral crepitation. Blowing in abnormal situation over hepatized lung. Decubitus, its significance. Course. Results. Favorable indications in pulse, breathing, face, temperature, appetite, decubitus, clearing of lung. Unfavorable indications in breathing, pulse, fever, face, uneasy movements pawing, cold limbs, prostration, nervousness, weakness. Sabacute Pneumonia. Terminations of pneumonia, death, resolution, splenization, abscess, gangrene, red hepatisation, grey hepatisation, fibrinous consolidation. Lesions: congestion, exudation and cell growth, hepatisation—red and grey, deliquescence, abscess. Blood, loss of red globules, increase of white, excess of fibrine, glandular swelling, pleurisy, degenerations in other organs, laminitis, rheumatism. Treatment: adapted to strength of subject and type of disease, hygienic, anti-rigor, antiphlogistic, expectant, stimulant, antipyretic, febrifuge, sedative, moist compresses, derivatives, laxatives. In subacute form tonics, heart stimulants, febrifuge. In chronic cases add rich digestible diet, and easy open air life.

This consists in inflammation of the spongy tissue of the lung involving mainly and primarily the walls of the alveoli and interlobular connective tissue with their respective trophic centres (nuclei). The acute congestion of excessive heart action and debilitated pulmonary capillaries described above, is primarily a disease of the blood vessels which become over distended and may or may not lead to the inflammatory processes in their walls and the tissues adjacent. Pneumonia on the other hand is essentially inflammation of these tissues and nuclei, with exudation usually of a fibrinous material into their substance.

PREDISPOSING CAUSES. 1. **Age.** A very early age is nearly exempt, and from six years upward there are fewer cases relative to the equine population, variations that may be better accounted for by stabling, training and acquired immunity than by the mere fact of age. Of 237 cases, 2 were $3\frac{1}{2}$ years; 32 were 4 years; 19 were 5 years; 131 were 6 to 11 years; 46 were over 11 years; and 7 of uncertain age (Trasbot). 2. **Sex.** No visible effect. 3. **Stabling, training, change of food.** While the young colt at pasture is practically immune, the period of stabling, transition to a dry and grain diet, and to the nervous excitement attendant on training and unwonted work, as shown in the statistics of Percivall and Trasbot, determine an enormous increase of cases. In a cavalry regiment, Percivall found that 56.6 per cent. of all lung diseases occurred before the fifth year, and Trasbot found that at the Alfort Veterinary College 13.5 per cent. of all equine pneumonias occurred in the fourth year. 4. **Hot Stables. Impure Air.** These two conditions usually co-exist and prove potent causes, especially in young horses brought from the fields. We cannot, however, separate this cause as usually observed from the action of pathogenic germs which are preserved and concentrated in such places. 5. **Poor Health.** Debilitating diseases, insufficient and poor diet, overwork, exposure to cold draughts or darkness and any other cause which lowers the vitality predisposes. 6. **A Previous Attack.** This usually leaves some structural or functional change which renders the lung more susceptible to a subsequent invasion. Against this must be placed the immunity which follows the contagious forms, but as this is usually exhausted in the course of six months it does not invalidate the position that the permanent impairment of pulmonary integrity is a predisposing cause. 7. **Plethora.** Tending as this does to congestion it must be accepted also for the next pathological step—pneumonia. 8. **Climate and Season.** This is notoriously an important factor. At Paris, Trasbot met with 237 cases in the nine months from October to June inclusive, and but eight cases in the summer months—July, August and September. In Great Britain, where the vicissitudes are less severe, Percivall had in the cavalry horses in the seven months, from October to April inclusive, 146 cases=20.85 per month, and in the five months, from May to September inclusive, 62 cases=12.4 per month.

Exciting Causes. Nearly all the above causes when acting with unusual force may become direct factors in causation. The effect of a sudden and extreme **chill** is especially to be feared. Even in cases that are unquestionably due to a microbe as the essential cause, the nervous disorder manifested in the chill, and the clogging of the pulmonary circulation in connection with the retrocession of blood from the surface of the body, furnishes the opportunity for the colonization of the germ. The average horse at pasture will stand with impunity cold storms of rain, snow, and sleet, and transitions from a warm noonday sun to a cold night-wind and dew and even frost, but under other conditions of the system, with the fatigue and fret and sudden changes of food and regimen attendant on domestication, or with any derangement of an important bodily function, the *chill* is often the manifest occasion of disturbance of the balance of health, and the supervention of pneumonia. **Fatigue**, a system charged with **leucomaines**, and a **free perspiration**, which is suddenly checked by exposure, at rest, to a cold rain or snow, to a draught **between door and window**, to **immersion in the cold waters** of a river, or to **sponging with cold water** is quite liable to cause pneumonia. An unduly heavy winter coat as an individual peculiarity or determined by a cold environment in autumn often predisposes strongly to such dangerous chills, by the frequency and profuseness of the perspirations and general relaxation of the system. Clipping of such subjects is a true hygienic measure though it entails the need of extra care in blanketing. Again in the animal that has already suffered from disease of the respiratory organs these chills are more dangerous factors.

Direct irritation by inhalation of smoke and other products of combustion ; or acrid or irritant gases or dust ; by the drawing of food by aspiration into the lungs (as in paralysis of the larynx or pharynx, choking, cough, apoplexy, vomiting, etc.) ; by pouring irritant or insoluble drugs (oil, lard) through the nose ; by the pressure of neoplasms (actinomycosis, tubercle, glanders, cancer) ; or by the presence of parasites (strongyles, distomata, echinococci, linguatulas, is another factor.

Pneumonia from **Contusion** of the chest, fracture of a rib, or puncture or laceration of the lung is recognized.

Contagion. The presence of a *contagium* in pneumonia is to-

day well established. Clinical observation had indicated this even before the discovery of a specific germ, but bacteriological investigations and the transmission of the disease by inoculation of artificial cultures have definitely settled the question. It does not follow that all cases are contagious, nor equally so, but the recognition of the contagious form satisfactorily explains the prevalence of the disease in one stable while an adjoining one escapes, and the eruption of new cases in a stable after an animal affected with the disease or convalescent from it has been introduced. It has been objected that many horses stand in the stable with pneumonia cases and escape, but so is it with glanders, cow-pox, and many other affections. It merely argues an immunity in the case of some, and for the disease germ a very limited transmissibility through the air. The further objection that the existence of lesions in the lung before the onset of fever, excludes this from the list of infectious diseases, is untenable since many undeniably contagious diseases, like cutaneous anthrax, glanders, lung plague, cow-pox, appear locally before any constitutional disturbance occurs, which comes on later as the result of extensive local disease and the circulation of toxins and microbes in the blood. It places contagious pneumonia however in that long list of infectious diseases which develop first locally in the seat of infection and later become more or less generalized.

It must be admitted, however, that the germ of pneumonia is not the same for all cases of the disease and for all genera of animals. It must also be allowed that the same germ does not always maintain the same degree of virulence, and that it may even live for a time on the buccal mucosa of an animal belonging to a susceptible genus without any morbid result. In short we must recognize that different germs of pneumonia may become temporarily non-virulent or only slightly virulent, and remain pathologically quiescent, as for example during the summer months, to reassert themselves later when the conditions become more favorable to pathogenesis.

BACTERIOLOGY.

BACILLUS OF FRIEDLÄNDER. This is a short rod with rounded ends, often merely oval, occurring in pairs or chains of

four and under given circumstances surrounded by a transparent gelatinous capsule. It is ærobic, non-motile, does not liquefy gelatine, nor sporulate, and in gelatine stick cultures has a nail-like growth. This was found by Friedländer, Frobenius, Weichselbaum and Wolf in the pulmonary alveoli in a small proportion of cases of fibrinous pneumonia in man. The cultures, injected into the lungs of animals, killed one dog (out of five), six Guinea-pigs (out of eleven), and thirty-two mice (all the injected). Lesions were intense congestion of the lungs, seropurulent pleural effusion, and enlarged spleen, while the bacillus swarmed in the blood and exudate.

MICROCOCCLUS PNEUMONIÆ CROUPOSÆ. First found by Sternberg in his own saliva in health, and by Pasteur in the saliva of a rabid child. Afterward found in the great majority of lungs affected with fibrinous pneumonia in man, by Talamon, Salvioli, Sternberg, Fränkel, Weichselbaum, Netter, Gamaleia, etc. Later it was found in meningitis, in ulcerative endocarditis, in arthritis, in otitis media, and in acute abscess in man.

It is a *spherical* or *oval coccus*, arranged in pairs, in fours, or exceptionally in eights or tens. Lanceolate forms (*streptococcus lanceolatus*) are the rule in the blood of animals, and circular in artificial cultures. It stains readily in aniline colors and by Gram's method, grows in ordinary culture media, at 37° C. in the absence of free acid, and in gelatine stick cultures, as small, white colonies along the line of culture without liquefying the gelatine. It dies in ten minutes at 52° C. (Sternberg). Its virulence lessens in artificial cultures, but is restored by passing through the body of a susceptible animal.

Injection into the lungs or trachea of rabbits, mice, sheep and, less certainly, Guinea-pigs, produced distinct fibrinous pneumonia filled with the microbe. In dogs, subcutaneously, it caused abscess, but in the lungs an acute fibrinous pneumonia which only exceptionally proved fatal, recovery usually taking place in ten to fifteen days.

Klemperer induced immunity, sometimes lasting six months, by intravenous injection of filtered cultures.

DIPLOCOCCUS (streptococcus) PNEUMONIÆ CONTAGIOSA EQUINA. First found by Schütz in the lungs of pneumonic horses in 1887. It is an oval coccus arranged usually in pairs or

in threes or fours, and surrounded by a transparent envelope. It stains in aniline colors but not by Gram's method. It is *æro-bic* and grows in gelatine at ordinary temperature without liquefying it and in sick cultures, forms a line of small, white, separate colonies which do not coalesce by growth. Does not grow on the surface of the gelatine around the puncture. Line cultures on agar are in colonies like minute transparent droplets. In bouillon it develops long chains.

Inoculated on the rabbit, Guinea-pig and mouse, it produced death with pneumonic affections (*hæmorrhagic* congestion or inflammation), but in some of the rabbits and Guinea-pigs it failed to take. Chickens and pigs proved immune. Injected into the horse's lung or as spray into the trachea it produced true fibrinous pneumonia. Fiedaler and others obtained similar results, Peter has found the *fæces* of pneumonic horses virulent, an important point in connection with disinfection.

Schütz found that 20 grammes of the culture, in an equal quantity of boiled water, injected into the horse's trachea, produced a rise of temperature by two or three degrees, with rigors, cough, accelerated pulse, elevated temperature, dyspnoea and prostration, but that this subsided in a few hours. By repeating this every thirty-six hours, the fourth or fifth dose would fail to produce a reaction and the subject proved immune. (See *Contagious Pneumonia*, Vol. IV).

CADÉAC'S *DIPLOCOCCUS PNEUMONIÆ EQUINA*. In the lungs of cases of contagious pleuro-pneumonia of the horse Cadeac found a round non-capsulated coccus appearing in pairs, or rarely in chains, and staining by Gram's method. It grew slowly in bouillon and agar at 37° C., forming on the latter, in twenty-four hours, a thick, whitish, oily drop, which, as it grew larger, assumed a silvery whiteness, and dried in the center. In bouillon it precipitated a powdery sediment. The reaction of the culture medium was unchanged. It lost virulence rapidly in artificial cultures or by a heat of 50° C., and it died in ten minutes at a temperature of 60° C. Virulence was long retained when dried, or even in putrid material.

This proved infecting to the ass, rabbit and Guinea-pig, while the cat and white rat proved immune. Intratracheal injection of the dog produced a transient pneumonia. The ass inoculated

with the blood of the infected rabbit died in three days, with a hepatized lung, pleurisy, and swarms of the microbes in the lungs, blood and internal organs. Rabbits injected intravenously had enlarged spleen, reddish exudate in the serous cavities, urine stained with hæmoglobin, and lungs and kidneys congested. With intratracheal injections the lesions were exclusively pulmonary. The pulmonary lesions were less constant in the Guinea-pig. Weakened virus caused pulmonary lesions only, without septicæmia. Probably identical with Schütz's germ.

It has been suggested that this coccus is at least closely related to that of pneumo-enteritis of the horse.

COCCO-BACILLUS OF LIGNIERES. Lignieres found in the exudation in the tissues in the early stages of contagious pneumonia, influenza, and strangles, alike, a small cocco-bacillus, ovoid, taking polar stains especially, bleaching in Gram's or Wiegert's (iodine) solutions, non-motile, non-sporulating, and non-liquefying. These are the characteristics of the microbes of septicæmia hæmorrhagica, classed together as *Pasteurella* by Trevisan, and producing in all cases very similar lesions. They are to be found in pneumonia only in the earlier stages, up to the eighth, or at most the fifteenth day, and though the pure cultures proved fatal to inoculated solipeds, cattle, sheep, pigs, rodents, and birds, these inoculated cases have not shown the extremely infectious qualities, which are such marked features of strangles, contagious pneumonia and influenza. Further, the acceptance of Lignieres' cocco-bacillus as the common cause of these diseases is in direct contradiction of all past experience of these diseases which shows a constant succession of cases of strangles, influenza or contagious pneumonia in any given epizootic, and not a complex mixture of these three affections as would happen if they had one common cause. The cocco-bacillus therefore can only be considered as a predisposing or accessory cause, while the real pathogenic agent which gives each disease its individual characters, is the microbe which is found associated with or succeeding the organism of Lignieres.

In addition we must recognize the *aspergillus pneumonycosis* (Vol. V) and the microbes which become pathogenic and maintain pneumonia as they do bronchitis.

Symptoms. The onset of pneumonia is not often seen by the

veterinarian, who is called in only after the cough, loss of appetite, hurried breathing and rigor have revealed illness to the attendants. Hence perhaps chill and rise of temperature have been placed among the earliest symptoms. The symptoms are more violent in the racer, trotter and other nervous animals. Trasbot positively claims, that considerable pulmonary inflammation and even exudation have taken place before there is any chill or rise of temperature. This is especially the case in the heavy lymphatic races of draught houses, which often, according to this author, perform their usual work for days after inflammatory exudation has set in. A fair counterpart of this is found in lung plague of cattle and it would indicate that both start from a local infection, which gradually extends until the systemic derangement is induced. As usually seen, and especially when it follows exposure to severe cold, a staring coat or a shivering fit ushers in the disease, the degree of the chill bearing some ratio to the coldness of the air and to the future severity of the malady. This may be accompanied by a small, dry cough, but without any other marked sign of lung disease. With the access of the hot stage the characteristic symptoms of lung disease are manifested, at first resembling those of *congested lungs*, but less severe than those given under that head. There is a distinct increase of the body temperature; the visible mucous membranes are suffused with a blush; the expired air feels hot upon the hand; the breathing, 30 to 40 per minute, is short and accompanied by much lifting of the flanks (*labored*); the cough is deep as if coming from the depth of the chest, but not so hard nor so painful as in bronchitis; the fore-legs are placed apart, the elbows turned out and the head protruded to facilitate breathing; the nose is turned to an open door or window if any such is available; the contraction of the muscles of the face, the dilated nostrils and the retracted angle of the mouth give an anxious expression to the countenance; the eyes are semi-closed; the pulse full but soft (*oppressed*), beats from 48 to 70 per minute; the bowels are slightly costive, the urine scanty and high colored; the skin inelastic—*hide-bound*—harsh and dry, though sweats may bedew it in parts; the loins insensible to pinching; and if there is any discharge from the nose it consists only in a reddish—*rusty*—colored mucus.

Auscultation and **percussion** complete the diagnosis. At the outset the inflamed portion of lung, usually near its lower part, conveys a *crepitating* sound to the ear, but as consolidation extends, the healthy murmur and the crepitating râle are alike suppressed over the whole extent of the hepatized portion, around the margin of which a line of crepitation betrays the limit of the advancing inflammation. A similar line of crepitation encircles the hepatized mass even when the exuded products are being absorbed and when the lung is being cleared up and restored to its healthy state. Thus the advance of the inflammation, and the progress of recovery can be equally followed by the crepitation which, in the different circumstances, betokens advancing inflammation or active absorption. When both lungs are involved the posterior parts are chiefly implicated, while if the pneumonia is single it may attack the anterior, median or posterior part, or the entire lung may become consolidated. If hepatization exists in the anterior part of the lung the thick fleshy shoulder will forbid any satisfactory examination, but if in the middle portion only, while the respiratory murmur is lost it will be replaced by a strong blowing sound (bronchial respiration) because the noise of the air rushing through the larger bronchial tubes to the posterior healthy part of the lung is conveyed with greater force to the ear through the consolidated lung tissue. This is audible from the lower third of the chest to the upper limit of hepatization. The respiratory murmur in the unaffected lung is always louder than is natural.

Percussion confirms these results. Over the hepatized lung where no respiratory sound remains, a dull, dead sound only is brought out by the impulse of the fingers or closed fist, comparable to that obtained by percussion over the muscular masses of the shoulder or haunch, and forming a marked contrast to that obtained over the surrounding healthy lung. There is not that tenderness on pressure in the intercostal spaces which characterises pleurisy, but a sharp blow with the closed fist leads to wincing and usually grunting because of the concussion to which the diseased part is subjected. By encreasing the force of such blows the deepest parts of the lungs, may be tested, since in this way dullness due to consolidation of the deeper portions of the lungs may be detected even though the superficial investing parts are healthy.

The nature of the symptoms will vary according to the extent and character of the inflammation, from mild febrile reaction, with excited breathing and slight crepitation, to the more severe varieties in which the intensity of the symptoms are such as to threaten suffocation.

A marked feature of pneumonia in solipeds is that the patient obstinately stands in one position and never lies down so long as the severity of the inflammation lasts. The sharp crest on the lower border of his breast bone compels the horse to lie on his side, and since in this position the whole weight of the body has to be overcome in any full dilatation of the chest, he cannot retain the recumbent posture when any serious impediment to breathing exists. Hence it is that the fact of a horse suffering from pneumonia having lain down and remained so for some time is justly accepted as an indication of improvement.

Progress and results of the disease. The general symptoms above noted, remain with more or less intensity throughout. After the first flush of heat, on the occurrence of febrile reaction, the limbs become alternately hot and cold, and in this the general surface partakes to a less extent.

The tendency of pneumonia is to a crisis and recovery. Certain days have been supposed to be critical and on the whole the third, seventh, eleventh and fourteenth are those on which a favorable change is most probable.

Among the more *favorable indications* are the manifest abatement of the high bodily temperature and febrile symptoms generally, the increasing ease and regularity of the breathing, the greater force, distinctness and slowness of the pulse, the permanent return of warmth to the limbs, the softer and more elastic feeling of the skin, the recovery of appetite, and above all, the turning of the nose from the open window or the retention of the recumbent position for a length of time. These symptoms will become more patent day by day, and the absorption of the effused products and the clearing up of the lung may be traced by the gradually decreasing area of dullness and of the circular line of crepitation as ascertained by percussion and auscultation.

If on the contrary the disease takes an *unfavorable* turn, some such signs as the following will manifest it : increasing rapidity and embarrassment of the breathing ; smallness and indistinct-

ness of the pulse, which is increased to perhaps 100 beats per minute ; tumultuous heart's action, the impulse of which is strongly felt behind the left elbow ; a more laborious working of the flanks ; frequent despondent looking toward the flanks ; pawing with the fore feet, lying down, and as suddenly rising again ; permanent coldness of the extremities ; hanging head with great dullness and despondency of expression ; dull, sunken, lusterless eye ; hanging lower lip ; leaden hue of the nasal mucous membrane ; convulsive twitching of the muscles of the surface ; reeling in gait, and extension of the crepitation to the limits of all the still pervious lung.

SUBACUTE PNEUMONIA. This term is employed to designate that subdued or milder form of the disease which sometimes arises independently and at others follows the acute.

In this variety the characteristic symptoms may be much less marked and the disease is less easily recognized. There is some acceleration and quickness of pulse, lifting of the flanks and heat of the mouth and body generally. There are alternations of heat and cold of the surface and extremities, a rough, unthrifty coat, hide-bound, a dull, listless moping manner and the same symptoms on auscultation and percussion as in the acute form.

The changes take place slowly, but the disease may prove obstinate and is often followed by permanent alterations in the lungs. Rheumatic affections of the limbs, inflammation of the feet, and other diseases frequently supervene during the course of this form of the affection.

The terminations of pneumonia are : by death ; resolution with absorption of exuded products ; splenisation ; abscess ; gangrene ; permanent consolidation with organization of exuded products. The disease will sometimes lapse into the chronic form.

Death is fortunately the least frequent issue. It may follow on rapidly advancing and general congestion of the lung—asphyxia ; from heart failure, the overworked organ becoming exhausted under the strain of forcing the blood through the virtually impervious lungs ; from hyperthermia, the limit of bodily temperature 108° F. having been reached or exceeded ; or from collapse or exhaustion.

In **resolution**, which is the most favorable termination, the

febrile and other symptoms subside and the exudations in the effused lung undergo a process of liquefaction and absorption until neither auscultation nor percussion nor even the examination of the lung after death will show the slightest trace of the pre-existent disease. This is the most common termination in single pneumonia in the horse.

Splenisation is that condition of lung already described under the head of *pulmonary congestion*, and if affecting both lungs throughout, necessarily destroys life by arresting the aeration of the blood.

Abscess. Diffuse suppuration is very common in the stage of gray hepatization. In this the affected lung becomes more or less extensively infiltrated with pus, limited by no distinct membrane like the pus of an abscess, but exuding freely from the cut surface of the lung or escaping from its interstices when it is pressed. It is preceded and in its early stages associated with the formation of granular masses and corpuscles. Its existence cannot be certainly ascertained though it may be surmised when after hepatization of a portion of lung a *mucous râle*, a sort of gurgling, is heard in the adjacent bronchium and an abundant muco-purulent discharge takes place from the nose. It threatens extensive destruction of lung tissue.

Circumscribed suppuration or *abscess* is infrequent though occasionally met with in the horse or ox. In this case the excessive exudation at one point liquefies, and the surrounding lymph becoming organized into a vascular membrane, an abscess is formed. This may burst into the bronchial tubes and be discharged by the nose. In less favorable cases it makes its way toward the pleural surface and opens into the cavity of the chest. It is impossible to detect the existence of a pulmonary abscess, though after it has burst into a bronchial tube the existence of the cavity may be ascertained by the amphoric sound heard on auscultation.

Animals may recover from such pulmonary suppurations, or if they are too extensive, the consequent depletion may induce hectic and death.

Gangrene of the lung is happily rare and has appeared to be connected with close, foul stables, previous ill-health, and work after the onset of pneumonia. It is characterized by high tem-

perature (106° to 108° F.) by great dullness and prostration, due to the poisoning of the nerve centres, by weakness and unsteadiness, by complete loss of appetite, and at length an intolerable foetor of the breath as if from putrefying animal matter. In rare cases recovery may take place, the dead portion having become detached and expectorated.

Consolidation from hepatization is the condition in which the inflamed lung is always found, in the second stage of the disease. The lung has then the density and brownish red appearance naturally belonging to the liver (*red hepatization*), which changes on the occurrence of softening of the exuded products to a grayish hue (*gray hepatization*). But after the subsidence of the acute symptoms, the process of liquefaction and absorption is not always complete, a portion of the exuded product becomes vascular, is developed into fibrous tissue and remains permanently impervious to air. Such is the state of the lung in many cases of *thick* or *short wind* in horses, when these have occurred as a sequel of pneumonia. A horse suffering in this way has the breathing habitually accelerated, and is thrown into a state of great distress by any attempt to make him perform hard work, such as galloping, dragging a load up hill and the like. A *chronic cough* may also accompany this condition.

Pathological Lesions. These differ according to the stage of the disease. In the *first stage*, that of congestion, the lung tissue is engorged with blood as described under the head of **congested lungs**. As early as 6 or 7 hours after artificial irritation, the alveoli of the affected part are already filled by exudation and cell proliferation. Until this has taken place the alveoli can still be distended by blowing into the bronchical tube.

In the **second stage** the condition of the lung is that of **red hepatization**, so called from its resemblance in color and consistency to the liver. There are gradations between congestion and *red hepatization*. In the earlier stages of the latter, the lung retains a measure of its softness, elasticity and permeability to air, though it is considerably firmer and less permeable than that which is in a state of congestion, and differs further from it in exuding from its cut surface not a grumous, dark bloody pulp, but a clear straw colored fluid. In the advanced *red hepatization* the lung is of a firm consistency and granular liver-like appear-

ance. In color it varies from a bright red to dark liver hue, the darker shades being chiefly met with in old animals or when the inflammation and fever have been intense and prostrating. Varying shades are seen in different lobules of the same lung. Its air cells are no longer pervious, it no longer crepitates under the pressure of the finger, nor floats in water, and its friability is such that it breaks down readily when the finger is thrust into its substance. Its surface is distinctly granular from the fibrinous plugging of the alveoli. Such a lung does not collapse when the chest is opened but retains its bulk and shape, and in some cases the diseased portion may, by reason of the abundance of the exudation, be really larger than the same portion of lung in a normal state of dilation. Its surface may thus retain the imprint of the ribs. Owing to the stasis of the blood in the vessels, a hepatized portion of lung cannot be injected. The exudation which infiltrates the lung tissue and obliterates the air cells, contains in the vicinity of the bloodvessels, numerous granular masses and corpuscles, and in the darker colored portions blood globules, owing to the diapedesis of the red cells, and the rupture of minute vessels. The smaller bronchial tubes stand out white and empty, showing that they have escaped the inflammatory action. Hepatization usually extends from the anterior lobe or lower border upward.

Gray hepatization is a sequel of the *red*, and at first presents the same firmness, friability and usually the same granular aspect; the lack of crepitation on pressure, and the higher density than water. From the cut surface a fatty or purulent fluid exudes spontaneously, or in other cases, only when pressure is applied. The granular masses and corpuscles have disappeared, and if supuration is not so abundant as to prove extensively destructive to lung tissue, that is gradually cleared up and restored to health. This state is always a very perilous one.

Abscess of the lung, sometimes met with in animals dying of pneumonia, shows a circumscribed area of inflammation and induration with the liquid pus in the centre, immediately surrounded by a vascular (limiting) membrane. Abscess may be single or multiple, though in the latter case it is commonly a symptom of pyæmia.

In *gangrene of the lung* the part may be in the dried condition of an eschar; it may indicate gangrene only by its altered color,

its flaccidity, its fetid smell and the altered appearance of all its microscopic elements ; it may be denoted by a putrid softening, the tissue easily breaking down into a stinking pulp of mixed fibrous and granular materials ; or lastly there may be merely a cavity with traces of putrid contents, the dead mass having been detached, disintegrated and expectorated.

Modifications of the Blood and Distant Organs.

A marked feature of pneumonia is the destruction of red blood globules. This is early indicated in the staining of the visible mucosa by the liberated hæmaglobin, and by actual count they may be reduced in the horse from 7,500,000 to 6,000,000 per cubic millimeter (Trasbot). There is an increase of white globules, an absolute increase, not only in ratio to the red. The hæmatoblasts are enormously increased especially during defervescence. The fibrine (fibrine formers) is materially increased ; in the horse from 3.5 to 6.7 or 7.5 per 1,000 (Grehaut). Albumen is diminished. Soda salts are increased. The bronchial lymphatic glands are always congested, swollen and reddened, with some serous effusion. They may become the seat of inflammatory cell growth (embryonic tissue) or even of suppuration. The abscesses may open into the bronchia or pleura. These are especially to be dreaded from their tendency to implicate the inferior laryngeal nerve and induce roaring.

Pleurisy is inevitable when the inflammation reaches the surface of the lung, hence hydrothorax is often present. Pericarditis and hydropericardium are similarly met with. Endocarditis is occasionally present and may be traced to strain of the valves of the laboring heart, or to direct infection with the pneumonia microbe. Dilatation of the right ventricle is common as a result of the obstructed pulmonary circulation.

Fatty degeneration of the heart and congestions of the intestinal mucosa, liver, kidneys and spleen are further complications.

Finally laminitis and rheumatoid affections occur as complications.

Treatment. This must be adapted to the nature and condition of the subject and to the character of the disease. A horse in vigorous condition, or with an acute type of inflammation, may be greatly benefited by an actively depleting treatment, whereas to

the same animal in a low state of health, or during the prevalence of an epizootic form of the malady, depletion may be destruction. It is not sought here, as is so often done in the consideration of this disease, to ring the changes as to the probability of a change of type in disease, or a change of theory on the part of physicians having affected the practice of bloodletting. True to our primary purpose of rendering the work eminently practical, we shall first notice the general management applicable to all cases, then the treatment of the two great types of the disease, acute (sthenic), and subacute (asthenic), leaving to the enlightened judgment of the reader to apply an appropriately modified system to that large class of cases which occupy an intermediate position.

A pure airy box is first demanded, with the windows or doors toward the south, or at least not turned in the direction of the prevailing cold winds. The craving for pure air, so strikingly shown by the position which the animal assumes, ought never to be ignored nor neglected. We do not advocate the system of the late Professor Coleman who kept pneumonia patients in open sheds exposed to all vicissitudes of temperature winter or summer, and yet the fact that many recovered under such treatment, as well as under a more rigorous system, having been turned out into the open fields amidst frost and snow, ought to open the eyes of all to the incomparable value of fresh air in this disease. The box then must be dry, cool and airy but without a cold exposure and without draughts of cold air.

Next in importance to pure, cool air is the comfort of the patient. Any tendency to chill, shivering, staring coat, or coldness of the surface and extremities is to be counteracted as far as possible. One or more blankets according to the condition of the patient and the temperature of the atmosphere, are valuable, and for the same reason a hood may be put on. Coldness of the limbs is to be met by active rubbing with the hand or with wisps of dry hay, and then wrapping up loosely in flannel bandages. Some apply to the limbs ammonia and oil, spirits of turpentine, and other stimulants and thus by a powerful derivative action, obtain an alleviation of the lung symptoms. For the same reason a mustard poultice on the chest, or the hot wet rugs recommended for congested lungs, often prove valuable in the earlier stages. Large injections of warm water and the supply of warm gruels

are not to be neglected when they can be employed. Measures such as these directed to check any chill and render the circulation free and uniform in the skin and extremities, if adopted during the cold stages of the fever, will sometimes succeed in bringing about a resolution of the pulmonary congestion and warding off a threatened attack of pneumonia.

The diet should be of a non-stimulating and laxative kind. Bran mash, linseed, oatmeal, or other gruels, carrots, turnips, scalded hay, or green food, if at the proper season, should be given in small quantities so as not to satiate.

Antiphlogistic Treatment. Half a century ago bloodletting was considered the remedy *par excellence* for pneumonia and it seemed justified by the marked relief to breathing and pulse, which usually at once followed a free bleeding. In a short time, however, the fever would rise anew and the distressing symptoms reappear, which led the school of Broussais to repeat the bleeding, *coup sur coup*, as often as the exacerbation appeared. There was no respite for either age or condition, the debilitated city toiler, the babe at the breast, and man of eighty tottering into the grave, had alike to submit to the lancet, and when the oppressive symptoms returned, the blood had to flow anew. Broussais himself, however, recognized his error in his later life, and remarkably enough, his conversion was effected through veterinary practice. His two carriage horses were successively attacked by pneumonia; the first was treated by bleeding *coup sur coup* and recovered; the second was put under a more conservative treatment and also got well, but while the first remained soft, flabby, debilitated and susceptible for a length of time, the second was on convalescence, at once able to go into active work. The enormous abuse of bleeding, led to its more complete abandonment than would otherwise have been probable, and the contrast between the high mortality of cases treated by excessive bleeding, and the lower fatality in pneumonias treated without phlebotomy on the expectant (let alone) plan of Dietl, or the stimulating method of Todd, Bennett and others served to hasten its abandonment. Yet in blood-letting we have an instrument for good or evil, which is not to be judged on slight evidence. The mere lessening of the blood pressure is to be little considered, as it requires the abstraction of nearly one-third of the entire mass of blood to visibly affect this. The vas-

cular walls at once adapt themselves to the lessened amount. Nor is the mere lessening of the volume a vital point. After moderate bleeding, this is made up in a few hours; after severe bleeding in 24 to 48 hours. The loss of adult red globules is more lasting. Bleeding to the extent of one per cent. of the body weight may have the number restored in seven days. The young red globules though rapidly produced have individually less hæmoglobin, and they can convey less oxygen to the tissues. This should mean less oxidation, less heat, less waste, less urea, uric acid, hippuric acid and other poisonous products in the tissues. Yet Baur says that in anæmia there is a greater metabolism of proteids and more excretion of urea. How easy it is to blunder in looking from one single point of view. Again after bleeding there is a great relative increase of the various forms of white blood globules, most of them young and therefore with somewhat altered functions. The paucity of red globules and excess of white, are brought about by the pneumonia and independently of bleeding, so that it is difficult to say whether the phlebotomist is enhancing an evil, or helping a natural therapeusis. It seems hopeless to estimate the effects of these and other changes in the blood after bleeding, upon the metabolic processes of nutrition, secretion and sanguification. This digression has not been made to elucidate the results or the *modus operandi* of bleeding, but rather to illustrate the complexity of the problem involved, and to warn against broad and unwarranted generalization from insufficient premises.

Even today practitioners of the soundest judgment meet with a limited number of cases in which they resort to bleeding, with advantage. These occur mainly in strong, robust constitutions, in individuals accustomed to an invigorating, open air life, liberal diet and abundant exercise. Even in these this measure is chiefly resorted to, to relieve an acute pulmonary congestion, with a dangerous distension and over charging of a fatigued and over-worked heart. In short, the condition is one closely allied to acute congestion in which the value of bleeding is all but universally admitted. It is especially warranted early in the disease, though it may still be adopted with caution, in a similar condition which has supervened at a later stage. A strong pulse and bright red mucous membranes, are not as has been supposed, essential prerequisites to its employment. The mucosæ may be

pale, or more likely cyanotic, and the pulse small and weak, from the overcharging of the heart and its tendency to failure, and it is to relieve these conditions that we may adopt this most potent of all measures for securing a temporary lessening of the blood pressure in the right heart, and pulmonary circulation. Even the transient relief may allow this to right itself, and then less radical or dangerous measures may be relied on. Bleeding should very rarely be resorted to, save at the onset of the disease; extensive exudation into the lung tissue, strongly contra-indicates it; it cannot be safely employed in the very young or old, in weak or debilitated subjects, when the pneumonia has elapsed or supervened on another serious malady, or when occurring in an unhealthy district. Delafond met with a very high death-rate from bleeding in a damp undrained locality. When bleeding is permissible, the blood should be drawn from the jugular in a full stream, from a large orifice, the finger being placed upon the pulse, and the flow arrested as soon as the blood is felt to pass along the vessel in a fuller, freer current, and the breathing is seen to be relieved. It can rarely be repeated with profit or safety, and in the vast majority of cases, can be well dispensed with altogether.

Antipyretic Treatment. When the temperature runs dangerously high, a temporary use of antipyrin, acetanilid, phenacetin, or other potent antithermic remedy may be resorted to. But agents that so profoundly affect the heat centres, are not devoid of danger, and should not as a rule be continued after the dangerous excess of temperature has been overcome. They may be looked on as valuable to temporarily obviate an extreme danger, rather than as a form of regular treatment.

The modern resort of applying ice bags to the chest may be similarly disposed of. In very high fever they have been apparently beneficial, but the danger of chill or injurious reaction is so great that they must be employed with the greatest possible consideration and care.

Tallianine (ozonized terpene), an antipyretic, antiseptic and stimulant of polynuclear leucocytosis, is now largely used intravenously (10 to 50 cc. daily) with the best results.

Refrigerant Febrifuge. Neutral salts such as saltpeter in 2 drachm doses every six hours may be given in the drinking

water. These are valuable for their cooling and eliminating action, and possibly in counteracting the viscosity of the blood and exudations. Acetate of potash, bicarbonate of soda, iodide of potassium or muriate of ammonia, may be substituted.

Stimulants. In debilitated subjects, or with low fever or oppressed heart, the stimulating diuretics like sweet spirits of nitre or liquor of the acetate of ammonia are to be preferred, and this is especially the case during convalescence. They at once sustain the flagging heart and aid in the excretion of morbid products. Digitalis is often of great value in the same sense, and as a heart stimulant *nux vomica*.

When the circulation is weak or flagging, alcohol, ammonia and its salts, ether, etc., are often of value and they may even act directly on the pathogenic microbe. The inhalation of oxygen, or the solution of peroxide of hydrogen (2 ozs., 1 per cent sol.) given by the mouth has often an excellent effect.

Sedatives. Aconite has become too much of a domestic remedy, nevertheless it may be used with advantage in high fever with excited heart action, to moderate the circulation and relieve the breathing. Veratrum, hydrobromic acid, bromide of sodium or ammonium, or chloral hydrate may be used as alternates or substitutes.

Compresses. Fomentations. Poultices. No measure is safer nor more promising, especially in the early stages, than the poultice jacket or compress. A blanket wrung out of hot water is wrapped around the chest, covered with a thick dry one, and held firmly attached by elastic circingles. Or soaked cotton wool is applied and covered with a dry blanket or a rubber sheet. The more acute the inflammation the more valuable is this measure.

Derivatives. As a derivative the mustard pulp rubbed in and covered with thick paper or rubber is especially valuable. In one hour it may secure a free exudation and material relief to the breathing. It may be replaced by ammonia and oil, with or without a covering, by hot air or water, or by cantharides. This must, however, be used with judgment. In the early stage with a high type of inflammation and fever, the surface irritation may aggravate this through sympathy; in such cases therefore the severity of the inflammation should first be moderated before using an active counterirritant. In debilitated conditions too, with an

altered or depraved state of the blood, and during the prevalence of a low type of the disease, sloughing may ensue from incautious blistering.

The repetition of the blister is often useful, the healing process going on simultaneously in the blistered surface and the diseased lung,, by virtue of nervous sympathy.

To complete recovery, a course of vegetable tonics, such as gentian, nux vomica, calumba, may be given with iodide of potassium for a week or more. Constipation occurring during convalescence must always be corrected by food, (bran mash, linseed gruel), injections, or oleaginous, saline, or aloetic laxatives. The greatest care should be exercised to secure pure air, comfort, sunshine, good grooming and general hygiene, and to prevent over-exertion during convalescence.

In the *subacute* types of pneumonia, the fundamental difference in the treatment consists in the avoidance of all depressing remedies, and the employment of stimulants and a supporting diet from the beginning. Sweet spirits of nitre and liquor of the acetate of ammonia, carbonate of ammonia or salammoniac with digitalis and strychnia, may be used from the first. Vegetable tonics may be resorted to at an early stage, tallianine, peroxide of hydrogen, and when expectoration is established and the fever moderated, even mineral tonics may be employed. Nourishing gruels, mash, roots, green food, and scalded oats may be used in turn, to coax the appetite but not to satiate. In other respects the treatment is the same as for the acute. This form of the disease is liable to prove obstinate and persistent, and there appears to be a greater tendency to complications and so called metastasis, as enteritis, laminitis or rheumatoid affections of the back or limbs. These, when they occur, must be treated as if they had arisen in ordinary circumstances, having regard meanwhile to the remaining inflammation in the lungs, for that has not necessarily been quite superseded but only alleviated.

CHRONIC PNEUMONIA. This has been described, but, if uncomplicated by consumption, it appears to be usually only that consolidation of lung, due to the organization of exuded products into fibrous tissue, which occasionally forms a sequel of acute inflammation of the lungs. In such cases an access of circumscribed local congestion is liable to result from overexertion, or a

chronic state of irritation is maintained, attended with more or less fever, inappetence, mal-assimilation, and often in the long run, hectic, under which the animal is worn out. In such cases the chief indications are to avoid overwork or any undue strain upon the breathing organs, to support the patient by nourishing and easily digested food, and to control and remove any local irritation by measures indicated under the head of acute pneumonia.

FIBRINOUS PNEUMONIA IN THE OX.

Subacute in many cases. Effect of temperament and work. Acute form. Symptoms. Decubitus. Unfavorable symptoms. Prognosis. Suppuration frequent: indications. Gangrene. Coliquative Diarrhoea. Lesions. Comparison with those of lung plague. Tubercle. Treatment: bleeding, laxatives, refrigerant salts, derivatives, stimulants, tonics. Chronic form. Symptoms. Treatment.

In the large ruminants this disease tends more towards a sub-acute type than in the horse, and coming on insidiously from ordinary causes is liable to be confounded with the *contagious pleuro-pneumonia* of the bovine race. As in the horse, the nervous animals show more violent symptoms. It is rare in milch cows and in young cattle and more frequent in work-oxen.

In the *acute form* the symptoms mainly agree with those of the horse. There is the same shivering, followed by a hot stage, hyperthermia, the accelerated pulse, the short, quick, labored breathing, heaving flanks, cough frequent, deep, hacking, and easily excited, dilating nostrils, redness of the mucous membrane, and the same indications on auscultation and percussion, care being taken to obviate misconception of natural conditions in the chest of the ox. There is in addition a dry muzzle, tenderness of the back and breast bones and wincing when they are pinched between the fingers and thumb; suspension of the appetite and rumination, and in cows suppression of the secretion of milk; the mouth is often opened and tongue protruded to facilitate breathing, and in bad cases each expiration is accompanied by a moan or grunt. In many cases the ox can lie on his flattened breast-bone and maintain the breathing process, but when the disease is severe

he stands no less obstinately than the horse, his elbows turned out, his nose protruded and directed towards a window or other opening.

Among the unfavorable symptoms may be mentioned increasing anxiety and distress, a more oppressed breathing, the animal standing constantly in one position with legs apart, elbows turned out, his nose extremely raised, nostrils widely dilating, mouth open, tongue protruded, the expiratory grunt deep and prolonged, the cough infrequent and so weak as to be almost inaudible, being rather like a forced expiration, and the pulse rapid, feeble or imperceptible. The prognosis is favorable in moderate cases subjected to early treatment.

The termination by *suppuration* is more frequent than in the horse. The general symptoms are ameliorated, appetite and rumination return though they remain capricious and irregular, there remains the double action of the flanks, the dry, rough muzzle, the tense inelastic skin, frequently varying in temperature, the beast shivers at intervals, the cough is weak and often repeated, a yellowish, thick discharge takes place from the nose, weakness and emaciation advance and the animal dies in from twenty to thirty days.

Gangrene of the lung sometimes supervenes and is indicated by similar symptoms as in the horse. In severe and prolonged cases a violent fetid diarrhœa often supervenes and hastens a fatal result.

The *post mortem* lesions are similar to those of the horse. The cut surface of the hepatized lung, however, is divided into irregular red spaces by intersecting yellow lines—hence the name of *marbled lung*, from a supposed resemblance to that stone. The red spaces represent the pulmonary lobules and the whitish lines the surrounding areolar tissue, which, being especially abundant in ruminants and pigs, stands out prominently when infiltrated with the yellowish exudation. There is then nothing specific in this appearance as has been erroneously supposed, it is merely the result of the different structure of the lung in these animals and is always seen in the hepatized lung unless when from extravasation of blood into its substance the redness is rendered uniform. The amount of exudate into the interlobular tissue is, however, never so great as in lung plague.

The greater frequency of suppuration in the lung of the ox, as well as the greater tendency to tubercular deposit in prolonged cases are additional features in the diseased lungs.

Treatment. Blood-letting should be employed only with precautions, as in the horse. A saline laxative (1 lb. Epsom salts and $\frac{1}{2}$ lb. molasses) may be used with advantage and safety early in the disease, though in advanced stages it may sometimes prove dangerous from the tendency to diarrhœa. If constipation appears at a late stage, injection of warm water and a mild laxative (6 ounces sulphate of soda) only, should be given. The laxative may be followed by the same neutral salts and in the same doses as for the horse. Counterirritants are of equal value. A mustard poultice may be kept on for several hours, or a mixture in equal parts of oil of turpentine, ammonia, and olive oil may be actively rubbed over both sides of the chest and repeated daily until tender. In Denmark a prompt and efficient blister is made with 1 part of Croton oil and 10 parts each of sulphuric ether and spirits of wine. This is rubbed actively over the chest and washed off as soon as a sufficient effect has been produced. It must be carefully watched to prevent blemishing. A hot air jet or hot water may be substituted.

In the low types of the disease and during convalescence, stimulants and tonics are to be employed as recommended for the horse.

CHRONIC PNEUMONIA. Gellé describes a chronic form of this disease in cows. For about a month the patient became increasingly emaciated, there was a frequent, dry, weak cough, lifting of the flanks, and expiration double and accompanied by a moan. All these symptoms were aggravated by gentle exercise. Percussion gave a distinct crepitating râle. The pulse was weak and rapid, the mucous membranes red and tumid, skin dry, ears and horns cold, appetite small and capricious, rumination rare, excrements soft, and milk almost dried up. Tuberculin test is desirable.

The *treatment* is by diuretics with vegetable tonics and stimulants and active counterirritation over the chest. Gellé considers the disease as all but incurable unless active blistering is promptly employed so soon as the malady has assumed the chronic form and before extensive structural changes have taken place in the lungs.

FIBRINOUS PNEUMONIA IN SHEEP.

Causes : damp, cold soils, inclement weather, cold rains, hard driving, shearing or washing in cold weather, change to a cold climate, or from a warm barn, hot barns, heavy fleeces, sudden plethora. Symptoms : in congestive cases, in inflammatory. Treatment : preventive, hygienic, antiphlogistic, laxative, febrifuge, derivative.

This disease is not unfrequent in these animals, occurring enzootically in low, wet pastures ; or from cold storms of wind, sleet or drenching rains, particularly after hard driving, or shearing ; or from washing during inclement weather. Dressing with mercurial ointment in cases of *scab* is a frequent cause of pneumonia and death in Lincolnshire and various other English countries. Lastly M. Seron in Hurtrel d'Arboval's "*Dictionnaire*" describes its prevalence in *Seine-Inférieure* among low conditioned sheep subjected abruptly to a very nutritious diet. The hot buildings, heavy fleeces, and sudden plethora, appear to conduce to dangerous pulmonary congestions. The *symptoms* do not differ materially from those seen in the ox, except so far as they are modified by the fact that the disease often terminates fatally before hepatization has been established and the symptoms, and post mortem appearances are those of congestion and sanguineous engorgement of the lung, rather than of hepatization.

This engorged state of the lungs, has led Youatt and others to describe them erroneously as "gangrenous" and shepherds to name the disease "rot of the lights." The condition is that of acute congestion, or in some cases anthrax or emphysematous anthrax.

The *treatment* ought to be chiefly preventive and will consist in the avoidance of the causes above indicated.

When the disease has set in, fresh air, and general comfort, bleeding, if in the very earliest stages and in a strong patient, purging (3 ounces sulphate of soda and $\frac{1}{4}$ lb. treacle in warm gruel) and a free supply of nitre (about $\frac{1}{2}$ an ounce daily to each) in the water or gruel supplied, are the leading indications. As a counterirritant aqua ammonia acts well being sufficiently confined by the fleece. For anthrax see Vol. IV.

FIG. PNEUMONIA.

Symptoms : chill, burrowing, hot skin, cough, disturbed breathing, indications of exudation. Treatment : laxative, sedative, nauseant, febrifuge, wet jacket, blister.

Hogs are not exempt from this disease. They show the same symptoms of chill with hiding under the litter, followed by a hot stage, cough, hurried breathing, and (if the clothing of fat is not too thick) conclusive results on auscultation and percussion.

As *treatment*, bleeding from the ears and tail is sometimes resorted to with questionable benefit. A laxative of three ounces castor oil or three or four croton beans given in the food is of value. Tartar emetic in doses of $\frac{1}{4}$ grain and nitrate of potash in 10 grain doses should be shaken on the tongue at least four times daily to keep up a continued nausea and action on the urinary organs. The tartar emetic, so worthless in the larger animals, is of value in the pig and dog. A damp compress or blister may be used. The skin of the animal is difficult to blister, but by the use of the Danish croton liniment, mentioned for the ox, of hot water, or of a mixture of oil of turpentine and croton, 8 parts of the former and one part of the latter, a sufficient effect can usually be obtained. In any outbreak affecting many, Swine plague is to be expected. Vol. IV.

DOG. FIBRINOUS PNEUMONIA.

Breeds most liable. Causes : over-exertion, cold baths, clipping, exposure in cold, distemper. Symptoms : chill, fever, disturbed breathing, cold extremities, cough. Treatment : dietary, nursing, laxative, nauseant, febrifuge, moist jacket, mustard, stimulants, tonics, heart tonics and careful nutrition during convalescence.

This is a frequent affection in hounds. In hunting or coursing dogs, the causes are like those operating in the horse. The clipping of long haired dogs in inclement weather, swimming dogs in winter without afterwards drying or heating them by exercise, and shutting them out of doors at night, when accustomed to a warm dwelling, are occasional causes. It sometimes occurs epizootically and frequently supervenes during distemper.

The chief *symptoms* of chill, fever, and difficulty in breathing are as in other animals, while the results of auscultation and percussion are more satisfactory than in any other domestic animal. The dog sits on its haunches to facilitate breathing; his elbows turned out, his mouth open and his tongue protruded. Coldness of the ears and short quick cough are usually marked symptoms.

Treatment. The general care applicable to other animals is equally demanded here. The diet should consist of mild broths, or farinaceous foods with a little gravy if necessary to render them palatable.

Bleeding from the jugular has been recommended and may be admissible at the outset of the disease in a very few appropriate cases. If costiveness exists a tablespoonful of castor oil may be given (more or less according to the size of the animal), following this up by the tartar emetic, nitre, tallianine and sugar recommended for bronchitis. The poultice jacket is of great value. Mustard poultices may later be applied to the sides of the chest. Stimulants, tonics and nourishing diet may be required during convalescence, or when the disease assumes a low type.

FIBRINOUS PNEUMONIA IN FOWLS.

Pneumonia in birds. Causes : exposure, neglect, foul coops, hot, close houses, etc. Symptoms : erect plumage, drooping head, wings, and tail, dark comb, gaping, panting cough, crepitation. Treatment : hygienic, laxative, febrifuge.

In chickens exposure and neglect are alleged causes. Foul coops and the contrast between the warm building and cold outer air are justly blamed. Ruffled feathers, drooping head, dark colored comb and wattles, trailing wings, a disposition to gape, panting and cough are noticed. Under the wings and over the back, crepitations and dullness may be detected. The patient may take a teaspoonful of castor oil, and saltpetre or iodide of potassium may be given in the drinking water. In careful doses the other remedial measures may be attempted.

BRONCHO-PNEUMONIA IN SOLIPEDS.

Synonyms : Catarrhal pneumonia, lobular pneumonia, capillary bronchitis, bronchiolitis.

Definition. A circumscribed inflammation of the lungs, usually limited to one or a few connecting lobulettes at one place, often multiple, and usually the result of extension from the bronchioles.

Causes. This is virtually the same malady described under the name of capillary bronchitis which can hardly fail to become extended to the alveoli and lung. If capillary bronchitis does not speedily subside, therefore, it inevitably merges into broncho-pneumonia. Again as bronchitis is usually attended and maintained by abundant microbial invasion, and it may be by various species of organisms, so broncho-pneumonia is a microbial disease without any constant and definitely fixed, single microorganism as its essential cause. All the irritant causes which lay the system open to bronchitis become equally important factors in broncho-pneumonia, and all climate vicissitudes and other influences which operate to produce the first disease also tend to produce the other. Finally it may occur from inhalation of irritant food or other elements and their lodgement in given bronchial tubes ; or it may be the result of the localization of given infections (tuberculosis, contagious pneumonia) in which case the disease takes its name from the infection in question.

Prolonged decubitus on the side, as in acute laminitis, quittor, pricks with suppuration of the subcorneal tissue, penetrating wounds of the perforans, navicular pulley or coffin joint, acute rheumatism or arthritis and other affections that incapacitate the animal for standing, tends to produce hypostatic congestion of the lower lung, and this at once weakens the part and renders the stagnant liquid in the bronchioles and parenchyma an inviting culture medium for invading microbes. These microbes may enter by the trachea and bronchi, or they may be carried in the blood from the lesion in the affected limb and reaching the debilitated and susceptible bronchioles and their contained serum, they at once colonize there and in the mucosa, and bronchitis is established. If then the microbes have not started primary colonies

in the infiltrated lung tissue, from microörganisms carried there in the blood, they soon extend into it from the infected bronchia, and broncho-pneumonia is definitely established.

The more debilitated the patient has become, the more readily do such hypostatic congestions and exudations take place, and the greater the danger of infective invasion. This complication is therefore to be looked for especially in poorly nourished, overworked and exhausted animals, and after old standing and enfeebling diseases.

The common causes of pulmonary œdema have a similar tendency, hence insufficiency of the cardiac valves, dilatation of the heart, disease of the kidneys, or even simple anæmia may be important elements in causation.

Pressure by enlarged lymph glands, tumors, phlebitis, or other neoplasm on the pulmonary branches of the vagus nerve or the sympathetic, predisposes to such congestions and œdemas, and even the rigid contractions of tetanus may bring them about.

Symptoms. These are those of bronchitis with violent dyspnœa, wheezing, or grunting breathing, cyanotic mucosæ, and soon the existence of circumscribed lung areas which give out a dull, flat sound on percussion in place of the normal pulmonary resonance. Around such areas of flatness, there may often be detected a line of crepitation, and in the centre of the solid area the heart beats, bronchial blowing, or râles, or abdominal rumbling may be louder and clearer than over the same parts in health. These may be further complicated by other abnormal sounds, as the flapping sound of dried exudate, the amphoric sound of an open vomica, the crack pot sound of a similar cavity with a closed valvular orifice, etc. The expectoration tends to become more profuse and purulent than in fibrinous pneumonia, and though the early rusty color may be absent it may be yellowish and streaks of blood are not uncommon.

Temperature usually runs high (104° to 106° F.) and if the pneumonia is extensive there is much mental dullness and prostration caused partly by the circulation of imperfectly ærated blood and partly by the retention in the system of the waste products of metabolism, and of the large amount of narcotic toxins produced by the multiplying bacteria. The toxicity of

these microbial products is shown by the fact that the subcutaneous injection of the exudate speedily killed a Guinea-pig.

Diagnosis depends on the persistence of a capillary bronchitis, the abundance and purulent character of the discharge, the marked dyspnoea and dullness and the existence of limited lung areas devoid of their normal resonance, and having a peripheral crepitation.

Lesions. The most marked lesions are : 1st the congestion and exudation in the bronchioles, with swelling and desquamation of the epithelium ; and 2nd the infiltration or hepatization of limited areas of lung tissue, the boundaries of which are usually conterminous with a lobule or a group of lobules attached to a particular bronchitic tube. These consolidated areas are studded with numerous minute, dirty white, or yellowish foci, each representing a terminal bronchiole with its cluster of air sacs and cells overdistended by the exudate.

Course. The progress of broncho-pneumonia is variable and somewhat uncertain. If the disease is arrested early, there is a gradual subsidence of the high fever, of the phenomena of broncho-pneumonia, of dyspnoea and anorexia, and a steady improvement and convalescence follows. Though somewhat further advanced the pneumonia may undergo resolution, the exudate being absorbed, the symptoms gradually improve and a complete recovery may have taken place in two weeks. Under less happy conditions there may remain thickening of the bronchial mucosa and submucosa, the latter even becoming fibroid in undergoing organization, so that contraction of the affected bronchium, with wheezing, results ; the epithelium may remain swollen, proliferate rapidly and contribute to produce a chronic discharge, or even an ulcer ; the tube may remain blocked leading to collapse of the dependent part of the lung ; fibroid or other nodules may appear outside the bronchial walls, or swelling or even abscess in the lymph glands, keeping up a chronic bronchitis, and by pressure on the vagus, recurrent or sympathetic nerve, causing pulmonary congestion or laryngeal paralysis (roaring) ; or abscesses or vomicae may form in the lungs giving rise to profuse, chronic discharge and loss of condition, with, it may be, infection of the pleura, or secondary abscesses in others parts. In many cases extensive pulmonary emphysema

occurs, leading to dyspnoëic breathing, accompanied by chronic discharge and predisposing to subsequent attacks or aggravations of the broncho-pneumonia and even of hæmoptysis (Cadeac). In all such cases the disease of the lungs is indefinitely prolonged.

Treatment. *Prevention* should be sought by avoidance of the usual causes of bronchitis, and especially of chill. In the case of animals that refuse to stand, hypostatic congestion should be avoided by propping up the animal with straw bundles or bags so that he will rest on the side of the sternum and lower part of the ribs, instead of flat on the side. When he can stand without injury he may be put in a sling.

Curative treatment will not differ materially from that of bronchitis on one hand and fibrinous pneumonia on the other. The high temperature may be met by abundance of cool drinking water, by damp compresses to the chest, by alkaline diuretics, or more promptly still by acetanilid. Or tallianine 20 cc. intravenously. For elimination of toxins, potassium iodide or bicarbonate, or nitrate may be used. As expectorants ammonium chloride or carbonate or acetate, or inhalations of fumes of tar, carbolic acid, creolin, cresyl, creosote, oil of turpentine, eucalyptol. One or other of these expectorants may be given in the form of electuary made up with liquorice and honey and smeared on the back teeth. Counterirritants to the sides of the chest are often of value,—soap liniment, aqua ammonia and oil, mustard, cantharides.

Tracheal injections of various kinds have been tried, the least irritating is hydrogen peroxide, while iodine solutions (iodine 1 dram, iodide of potassium 5 drams, water 1½ quart) tend to prevent the propagation of invading microbes and check their ravages, but in certain susceptible subjects the iodine itself proves too irritating to the tender mucosa.

INFECTIOUS BRONCHO-PNEUMONIA OF CATTLE.

Synonyms. Corn-stalk disease.

History. This has been known for a length of time in America, as appearing more particularly among cattle shipped from the West, and sometimes attacking, at intervals, all or nearly all of a shipment of cattle that have been subjected to the same environment, but without showing the disposition to indefinite propagation as an epizootic, contagious disease, which characterizes lung plague. In common with the broncho-pneumonias developed by the foul air and overcrowding on the Atlantic steamers, it gained a great notoriety in Europe where it was at first mistaken for the genuine lung plague. In 1888, Billings reported it as the *corn-stalk disease*, attributing it to a bacteridian parasitism of the stalks of Indian corn. In France it was studied by several veterinarians in imported American cattle, and chiefly by Nocard, who made cultures of its microbe and inoculations with pure cultures, and definitely established the contention of W. Williams, in Scotland, that it was a form of broncho-pneumonia entirely distinct from lung plague.

Causes. The essential cause of this affection is a bacillus about $1\ \mu$ long by 0.3 to $0.4\ \mu$ thick, highly motile, easily stained in aniline colors, ærobic (facultative anærobic) and growing luxuriantly in buillion cultures, of a neutral or alkaline reaction. On peptonised gelatine, growth is slow and spare, producing a thin, transparent, bluish pellicle with deeply notched borders like a fern leaf, and without liquefaction of the gelatine. On gelose it grows luxuriantly. On potato it grows slowly and sparingly forming a delicate, grayish film which shades off insensibly at the borders. The germ has great vitality retaining its virulence for a length of time in artificial cultures.

Nocard inoculated the bacillus in pure artificial culture successfully on mice, rabbits, Guinea-pigs, pigeons, cattle and sheep.

Inoculations on chickens, dogs and cats were negative. Two or three drops in rodents or pigeons caused death in forty-eight hours, with intense congestion of the mucosæ generally, but without local lesion in the seat of inoculation. Injection into the peritoneum caused death in fifteen to eighteen hours with intense purulent peritonitis.

Sheep and calves taking 1 cubic centimeter subcutem, had high fever, temperature 105.8° F. with anorexia and great prostration from which they recovered in a few days. Similar inoculations into the lungs proved fatal in forty-eight hours with extensive broncho-pneumonia and pleurisy.

As the affection is caused by a microbe which enjoys a saprophytic existence outside the animal body, the same conditions favor its attack as in simple bronchitis: (chill, climatic vicissitude, low condition, fatigue, excitement, hot, close stables, the inhaled dust of railway travel, etc.). Yet the disease occurs in cattle at pasture where nothing appears as a factor except the microbe, and perhaps a chill, or an attack of costiveness and indigestion. Pneumonias due to different microbes are classed under this name, none of them being so actively infectious as lung plague. Out of its area of origin the disease rarely extends beyond the imported herd.

Symptoms. After an uncertain incubation the patient becomes dull, weary, separates from the herd, and has hurried, oppressed breathing, sometimes with a grunt, a short, hacking cough, anorexia, agalactia, costiveness and a remarkable rise of temperature (107° F. to 108° F.). The mucosæ assume a yellow tint and the urine becomes red as the result of destruction of the red globules and the diffusion of the coloring matter through the blood, tissues and urine. Auscultation and percussion bring out indications of the lobular infiltration. Percussion resonance is lost over small areas especially toward the lower part of the lungs, and around the margin of each flat area there may be detected a line of crepitation, while in the center of the area are heard sounds produced in other parts and abnormally clear for that situation (heart sounds, blowing and mucous râles, abdominal rumbling). The symptoms gradually increase in severity and death may supervene in from eight hours to eight days. In favorable cases the progress of the malady is slow and improvement may set in early and go on to a complete recovery. In some instances chronic lesions are left in the lungs which continue to interfere with respiration.

Diagnosis. The origin of the cattle in a locality where this disease is known to occur, the fact that they had the range of corn-fields, or were fed corn-stalks, the sudden development of

the disease, the very high temperature, the yellow mucosæ and blood-stained urine, associated with the oppressed breathing and physical signs of local consolidations of lung tissue form a picture which is almost pathognomonic. The occurrence of a number of cases in the same herd, with little disposition to extend to other adjacent herds having a different feeding and environment is still more conclusive. In lung plague, except in very hot weather, the disease develops much more slowly, and at an early stage the amount of lung infiltrated is much more extensive, while the indications of destruction of blood globules are virtually absent. In black-quarter, affecting the lung, there is liable to be an implication of the walls of the chest, and in such a case there is crepitation under the touch, and the exudate contains the sporulating round-ended bacillus in abundance. From the ordinary type of bovine hæmorrhagic septicaemia it is distinguished by the absence of the external exudations which so frequently characterize that disease.

Lesions. On opening the chest there is usually found some liquid exudate, and there may be adhesions between the lungs and the ribs, yet neither liquid nor false membrane is likely to be as extensive as in lung plague. The lungs, especially in the anterior lobes and in their lower portions, are filled with exudate in areas usually circumscribed by the margins of lobules. If the exudate is recent it is still liquid or semi-liquid, and the part retains much of its elasticity and tenacity. Viewed on the surface, the affected portion of the lung is marked off into islets, representing the lobulettes, by the lighter colored exudate in the interlobular connective tissue, and again in the centre of each ultimate lobulette a light colored area representing the infiltrated bronchiole and air sacs. Such portions, when incised, allow the escape of a considerable amount of blood and serous exudate. In parts where the morbid process is of older standing the exudate has coagulated, and the affected lung is solid, compact and friable, from a clear red to a dark brown or black, and on section it shows a marbling like that of lung plague, only the yellow lines of interlobular tissue are much less thick than in that complaint, and the hepatization tends to be more sharply circumscribed by the outline of individual lobulettes. The hardness and friability encreases toward the centre of the lobulette indicating the greater

activity of the morbid processes in and around the bronchioles. The walls of the bronchia show marked congestion and redness with infiltration of the mucosa and submucosa. The mucopurulent contents, filling the smaller bronchioles to repletion, and the exudate which drains from the recently affected lung, equally swarm with the specific bacillus, usually in pure culture. The abundance of this microbe serves to complete the diagnosis from lung plague, in which the exudate is remarkably clear and free from granules and visible microorganisms.

Prevention. As in all infective diseases the important consideration is to keep susceptible animals away from the source of infection, and as that attaches to certain localities and their products in this disease, cattle should be denied access to these. Infection from animal to animal should also be provided against, yet this is of less account, as the disease is largely enzootic, and though cases may be adduced in which contagion must be conceded, yet, out of its primal locality, it has never shown a tendency to spread without limit as do the epizootic lung plague and other contagious diseases. It is not necessary, therefore, to surround the infectious broncho-pneumonia of cattle with the elaborate and unbending system of safe-guards which are demanded in a true plague. The quarantine of the single infected herd will usually put a speedy end to the trouble.

SEPTIC PLEURO-PNEUMONIA IN RUMINANTS AND PIGS.

Synonyms. Pneumo-enteritis of the young, April disease of calves. Calf-disease. *Septicæmia hæmorrhagica*.

Definition. A bacteridian disease of young ruminants and swine, characterized by a lobular pneumonia, pleurisy and often enteritis. See Gastro-enteritis in Calves, Vol. II.

History. Reported by Roche Lubin in 1851, in France; it was noted in calves in 1884-5, by Castagna, Rodina, Griffa, and Perroncito in Italy,—in pigs in 1887 in Holland, by Poels,—and by Stohr, Ellenberger, Funstug, Trincherd, Semmer and others in Germany.

Causes. The essential cause is a microbe, *pneumo-bacillus septicus*, but which has been held to be polymorphic having been described by Perroncito as a *micrococcus*, and again by the same observer and Galtier as a *diplococcus*, and again by Poels and Semmer as a very fine *bacillus*. It belongs to the *hæmorrhagic septicæmia* group, and has the characters of Trevisan's *Pasteurella*:—a very small organism like that of mouse and rabbit septicæmia, showing polar staining so as to resemble a diplococcus, staining readily in anilin colors, and is bleached in iodine solutions. It is ærobic yet facultative anærobic, and grows readily in ordinary culture media, soil, water; manure, liquid manure, and forages, and survives drying, putrefaction, freezing, and the presence and growth of cryptogams. The contagion, therefore, may come through water, food, or soil, or by passing from one animal to another, as when an infected calf or pig is brought into a building. It may enter the system by the mouth, from the teats of the dam, the milk or food, the trough, the water, the dust, and by open sores, particularly the navel of the new born. In the last case it may remain quiescent for a length of time in a thrombus, to develop the disease later when the clot comes to be broken up. Intratracheal injection of cultures of the bacillus promptly produces the lung affection. Perroncito noted that outbreaks were especially common in the litters of sows, fed cow's milk.

The new born are most subject to the affection, the germs evidently entering through the umbilicus. After six weeks of age

the period of danger from infection has passed. Calves born in an infected building suffer, as do those brought from sound herds into such a building shortly after birth. If, however, the dam has been moved into a fresh, non-infected stable a few days before parturition, the progeny born there usually escapes (Trincherd).

Spoilt, impure food, foul, close air, crowding, and unclean stables doubtless contribute to its propagation, yet it will develop and persist in the most admirably appointed buildings in apparently the most abundant and purest air, and with food and water that appear to be above suspicion. Even a thorough purification and disinfection of the barn will sometimes fail to check it, so that we must suspect that it attaches for a time to the individual animal in certain cases. In this connection Galtier attributes a coincident abortion in the dams to the propagation of the same microbe, and in such cases the calves may be already diseased at birth.

Prognosis. The mortality varies from 20 to 25, and in some outbreaks up to 66 per cent. In the most fatal type occurring within one or two days after birth the mortality is still higher. (See Vol. II.)

Lesions. 1. *Calves.* In acute cases the pleura contains a reddish, yellowish or milky liquid which may be so abundant as to cause compression and collapse (atelectasis) of the lungs. The affected portion of the lung is filled with a liquid, giving a yellowish color and a great increase of thickness to the interlobular tissue as in cases of lung plague, the contrast being strongly marked between the red parenchyma, and the yellow interlobular tissue. In case of blood extravasation, however, the color may be uniform. The surfaces of the pleura and bronchial mucosa show more or less petechiæ. The coagulated exudate may bind the lung to the ribs. In the bronchia and trachea the exudate may be serous with loose coagula, and even streaks of blood, and patches of congestion and petechiæ may be seen on the laryngeal and nasal mucosa, and in other parts of the body. In more advanced and chronic cases, the lungs may be solidly hepatized or they may on section show gray hepatization, or caseation. The bronchial and mediastinal glands are greatly swollen and congested, of a dark red.

2. *Pigs.* The lesions are in the main as in calves: pleural effusions, with false membranes, infiltration of the lungs with the same yellow marbling as in calves, hepatization in circumscribed areas, surrounded by a zone of liquid infiltration, softening necrosis, caseation and petechial discoloration are often all noticeable in the same case. The areas representing the different successive stages of the disease, are in keeping with the frequent continuation of the disease in a subacute or chronic form. Pericarditis is a common accompaniment.

Symptoms. Calves. Within a few days after birth, usually within the first week, there sets in a high fever, temperature 105° to 107° F., with cough, hurried, labored breathing (40 to 60), rapid pulse (90 to 100), and wheezing. The cough is frequent, small, suffocating, and the visible mucosæ deeply congested. The calf stands with legs propped outward, and head extended, and staggers if made to walk. Percussion causes pain, and shows areas of flatness, representing the consolidated lung and over such points an abnormally loud blowing sound may be heard. The natural tendency is to a rapid advance, with increasing difficulty of breathing, frothing about the lips, open mouth, pendent tongue, moaning with each expiration, diarrhœa, and great loss of flesh and weight.

Symptoms. Pigs. The symptoms in young pigs resemble those of swine plague to which this is closely related. There is the high fever, hurried breathing, wheezing, cough, tenderness on handling or percussion, petechiation of the skin, snout, and mucosæ, weakness of the limbs and diarrhœa. Its most marked diagnostic symptom is the fact that the disease often confines itself to the young pigs, while entirely respecting the older ones.

Prevention. Avoid all the known causes of the disease. As the food and surroundings of the dam become subservient to the propagation of the germ to the young, the first care must be given to her. No food, whether soiled fodder or such as has come from an infected barn, or in the case of sows no cow's milk, that may be suspected, and no raw swill from hotel, boarding-house, nor family kitchen, no refuse of the butcher stall or abattoir should be allowed. Avoid all dusty hay or any food altered by cryptogams or bacteria. Let the feeding troughs and racks be frequently cleansed and deluged with boiling water and

copperas solutions. Stables and pens are best made of cement rather than the more porous wood, and should be well drained, kept clean, and frequently whitewashed. The free introduction of pure air without draughts is important. If the dam can be sponged all over with a disinfectant and removed to a fresh unused stable before parturition, the bag sponged with a solution of hyposulphite of soda (two ounces to the gallon), and not returned to the infected building for some weeks, nor attended nor milked by any one attending the animals in that building, the disease may be avoided. Washing the body of the young animal and particularly the naval, just after birth, with a solution of creolin will greatly assist. In the warm summer season the disease may often be cut short by keeping the dams and offspring in the open fields for the first few weeks, which constitute the period of danger. The manure should be disinfected and removed from the pens several times a day and fresh litter supplied, or the floor and litter should be freely sprinkled with a solution of creolin, copperas, or blue stone.

Any young animal affected must be instantly removed from its fellows and the pen disinfected. Each patient must be fed separately from a pail or feeding bottle retained for its own exclusive use. The udder or teats that it may have sucked must be carefully washed and sponged with a solution of one-half ounce hyposulphite of soda to a quart of water. If any milk has been yielded by the dam before sponging, it should be boiled before being fed. It is important to avoid undue distension of the stomach by too heavy a feed, as the resulting indigestion lessens the resistance to the invading germ.

The carcase of an animal which has died of the disease should be burned or deeply buried, all dejections thoroughly disinfected, and the pen cleaned and whitewashed. No affected animal should be allowed to enter on a public highway, unfenced place, field, yard or house lest the infecting discharges be left to inoculate others. Common drinking troughs or vessels are of course forbidden.

All manure from the infected stable or pen should be burned when feasible, or disinfected, or plowed under on ground to which the genus of animals supplying it can find no access.

Disinfection of the buildings and contents may be made by thorough cleaning, the removal of all rotten wood, the exposure

of hollow wooden walls by tearing off the boards on one side, and then applying thoroughly a whitewash made of recently burned caustic lime, adding to every gallon of the mixture $\frac{1}{4}$ lb. of chloride of lime. Copper sulphate (5:100) or sulphuric acid (2:100) are excellent disinfectants but do not show so well as the whitewash when any portion has been imperfectly done, or omitted altogether. Even after a thorough disinfection the disease may continue to appear in the same building, suggesting the presence of the germ in some of the cows, perhaps in the alimentary canal or genital passages, to plant the infection anew. In other cases the removal of the infected animals and the thorough disinfection of the building have put a prompt and permanent stop to the disease.

Treatment. Every effort should be made to limit the extension of the infection to vital organs, and as the blood in the umbilical vein forms an excellent culture medium to discharge fresh supplies of the microbe and its toxins into the system, it is sometimes possible to disinfect this in recent cases in which a small pledget of cotton on the end of a staff can still be introduced into the open end of that vessel. The pledget, soaked in a 5 per cent. solution of carbolic acid, is passed as far up toward the liver as the vein will admit and the infecting bloody contents removed.

Elimination by the bowels and kidneys has been sought by giving daily $1\frac{1}{2}$ oz. cream tartar and 1 dram bisulphite of soda to a calf. Or salicylic acid or salicylate of soda or even quinine may be employed. The stomach should not be overloaded by too heavy a feed.

Inhalations of oil of turpentine, sulphurous acid or other antiseptics can be tried, but with no great hope of success. Bass claims excellent results from injection into the trachea of the calf of $2\frac{1}{2}$ drams daily for eight days of a mixture of iodine, 1 part, iodide of potassium, 5 parts, and water 400 parts. For lambs and pigs $\frac{1}{2}$ dram of the same preparation may be used.

Treatment is, however, too often unsuccessful and therefore prevention must be our main object.

BRONCHO-PNEUMONIA FROM INHALATION.

Synonyms. Inhalation Pneumonia, Inhalation Bronchitis. Broncho-pneumonia from foreign bodies. Alimentary Pneumonia. All forms of broncho-pneumonia are due to the inhalation of infinitesimal bodies—microbes—but this article will deal rather with cases in which the disease is induced, or complicated by the introduction by way of the larynx or trachea of larger bodies, liquid or solid, which remain as mechanical irritants in the air passages. Such extraneous bodies, with some exceptions in which they have been sterilized, are invariably the bearers of septic germs, so that, to the mechanical injury there is superadded active fermentation in the foreign bodies themselves, in the bronchial walls and finally in the lungs, with local and it may be general poisoning by the septic products of the micro-organisms. All such cases are characterized by the visible presence of the foreign matters in the bronchia, usually in an advanced condition of putrefaction, by the foul odors, by the implication, *first*, of the mucosa with cloudy swelling and desquamation of the epithelium, and a serous exudate much altered by the septic products, *second*, by peribronchial, and interlobular, and lobular inflammation and infiltration.

INHALATION PNEUMONIA IN SOLIPEDS.

Causes. Excluding microbial invasion, the extension of pre-existing bronchitis by the deeper penetration of the muco-purulent products into the capillary tubes, and the effects of the inhalation of irritant gases, we have still a large class of cases determined by the presence of larger bodies of extraneous origin. *Liquid* inhalations occur most frequently from the forcible administration of medicines in a fluid state. The length of the soft palate in the horse in a measure protects him against this danger, as he can hold the liquids in the mouth indefinitely, refusing to swallow voluntarily. This is, however, met by the ill-considered ingenuity of man, who pours the fluid through a tube passing through the fauces, or manipulates the larynx violently pushing it up against the pharynx to rouse reflex deglutition, or closes the nostrils, or, worse still, pours the medicine in a constant stream through the nose. In the later case the animal

has no power of resistance and must swallow the liquid as fast as it reaches the pharynx. But unfortunately for him he must also breathe, and with each act of inspiration a portion of the continuous stream is inevitably drawn down into the lungs. The effect will be in proportion to the tenacious or irritant properties of the material inhaled. One of the blandest agents, melted lard, is especially dangerous because of its filling the bronchia, sticking obstinately to the mucosa and excluding the air. This may cause suffocation in a few minutes. The more irritant oils, castor oil, croton oil, whale oil, produce local inflammation by contact and absorption.

A less dangerous practice is to give the liquid medicine through the mouth and compel the patient to swallow it by pouring a few drops of pure water through the nose. When the latter reaches the pharynx deglutition is at once produced and the buccal contents are carried on in the natural way. But should any of the water entering by the nose drop into the glottis, a cough is at once produced with its succeeding sudden, deep inspiration and the medicinal liquid is liable to pass down the wrong way. If swallowing is forced by the introduction of water through the nose, it should be in a few drops only at a time, and not in quantity, nor continuously, nor when the patient is about to inspire.

The recumbent animal is very liable to inhale medicines administered in liquid form. Hence the accident is more frequent in colics and paralysis than in other affections. When lying down the stomach is compressed and does not readily admit the liquid. It may cause regurgitation from the gullet if not from the stomach and the liquid reaching the pharynx is liable to be drawn into the lungs.

Pure water introduced into the trachea and bronchia is comparatively harmless. Gohier's pupils poured thirty litres into the lungs in this way without effect, though they killed another horse with forty litres poured in a steady stream. Colin killed a horse by pouring into the lungs, in a full stream, 30½ litres, cold from the well. Respirations became 55 to 60 per minute, but after death the lungs showed only a slight infiltration toward their lower border, and there was no water left in the bronchia but only some reddish froth. Cadeac and Malet claim that a certain

amount of distilled water caused broncho-pneumonia, whereas the same amount of common city water had no ill effect. If there was no other cause for the trouble it would seem as if the salts and other solids in the city water, giving it a greater density, withheld it from causing the same exudation and inflammation. The microbes carried in from the mouth or pharynx may easily cause broncho-pneumonia.

Of other agents suspended in the water thrown into the lungs, some, that are in full solution or very volatile, may be quickly absorbed and do little or no harm.

A great number of different agents have been thrown into the bronchia and have disappeared by absorption without leaving any marked irritation. This is true of strychnia (Majende), solution of extract of nux vomica (Colin, Bouley), alcohol (Colin), compound solution of iodine (I. 1, KI. 5, H₂O. 400) (Dieckerhoff), opium, quinine, extracts of gentian and belladonna (Goubaux). The following agents have caused bronchitis or broncho-pneumonia :—ammonia, oil of turpentine (Cornil, Trasbot, Masalongo), iron chloride (Veragut), silver nitrate, extract of cantharides (Cornil), chloroform, nitrate of potash (6 grams in 500 water) (Cadeac), chloral (25 grams to 100 water) (Cadeac, Malet), ether (P. Bert) and soda sulphate (Laquerriere).

Insoluble bodies in suspension in water or watery fluids, or inhaled as dust remain as sources of irritation. Road dust from limestone, clay and, above all, fine sand, prove a source of great irritation and may lay the foundation of bronchitis, broncho-pneumonia or tuberculosis. The anthracosis pulmonum, coal-miner's lung, from inhaled coal dust, the chalasis pulmonum, stone-hewer's lung, and siderosis pulmonum or cutler's lung, from the fine particles of iron or iron oxide, are familiar examples of trade diseases in man due to inhalation of angular, irritating, insoluble materials. In cities burning soft coal, and subject to fogs, man and beast alike inhale carbon particles which accumulate in the air cells, lung tissue and bronchial glands.

Zboril records the case of a horse, at the Vienna Veterinary School, which showed in a bronchium a branch of Robinia pseudo-acacia (locust) 30 centimeters long and with four short branches feathering backward, so that it could pass forward but could not return. The bronchium was dilated and the adjacent lung was consolidated with its pleura adherent to the ribs.

A common cause of the inhaling of foreign bodies is found in injuries or paralysis of the central or peripheric nerves. Experimental section of the vagus nerve showed long ago a resulting congestion of the bronchia and lungs and the inhalation of foreign bodies. The latter depends usually on the motor paralysis of the larynx through implication of the recurrent laryngeal nerve. But the same result may follow injury to the superior laryngeal and the sensory paralysis of the larynx. Any wound, therefore, implicating the vagus in front of the base of the heart, or the superior or inferior laryngeal nerve may thus lay the foundation of broncho-pneumonia, and any inflammatory swelling or tumor along the course of these nerves may bring about the same condition. The phlegmons of the pharyngeal region in strangles, and the resulting abscesses, the collections in the guttural pouches, adenoma, actinomycosis, carcinoma, or other morbid formations in this region, phlebitis of the jugulars, and phlegmons, abscesses or tumors of the prepectoral or bronchial lymph glands, may one or other paralyze the vagus or its branches and impair the functions of the larynx with resulting inhalation.

Paralysis of the peripheral nerves conferring sensation on the mucosa of pharynx and larynx, is caused by solutions of tartar emetic, and certain styptic and astringent agents so that the pharyngeal and laryngeal muscles fail to respond to the presence of solids and liquids and these accordingly fall into the larynx and descend into the lungs (Bouley). On the same basis a local paralysis or anæsthesia might be expected from cocaine, ether, chloroform, chloral and other anæsthetics.

Central paralysis from brain disease and especially such as comes from trouble in the pons or medulla has the same effect. Among these may be named the paralysis from cryptogamic fodder, from vetches (*vicia sativa*), from ripening ryegrass (*lolium perenne*) from millet and other plants. Encephalitis, meningoencephalitis, hydrocephalus, and dourine come in the same list.

In advanced tetanus the extension of spasm to the pharynx and larynx, may lead to inhalation.

Finally, in the course of surgical operations under an anæsthetic, inhalation of food material into the lungs will occasionally take place. If the operation is begun while the stomach is full, digestion may be partially arrested and some gastric tympany in-

duced which tends to efface the folds of mucosa covering the cardia. Under the struggles and compression of the abdomen some of the contents of the stomach are forced into the œsophagus and carried up to the pharynx, which being more or less unable to perform its functions the food materials drop into the larynx and are drawn down into the lungs. It may be added that during a prolonged operation, with the animal prostrate on its side, there is liable to be hypostatic congestion of the lung which lowers the resisting power, and causes it to readily succumb to the irritation caused by the foreign bodies and the invasion of the attendant microbes. Hence inhalation pneumonia after an operation often assumes a dangerous development. Again, in an operation on nose or throat, the blood may pass into the lungs in quantity.

Lesions. Both lungs are usually affected, though as a rule, neither is involved throughout. The centres of inflammation are multiple, and the boundary of each diseased area usually corresponds to the limits of a lobulette or group of lobulettes clustered around the termination of the bronchium which was blocked or primarily affected at that part. The individual lesion represents a broncho-pneumonia in which the inflammation in the lung tissue proper has been consecutive to the bronchitis.

On the surface of the lung when exposed, these inflamed lobulettes stand out above the level of the sound lung, red, firm, airless, noncrepitant and not collapsible. The inflamed areas are most commonly in the anterior part of the lung near its lower border, but may be at any point. They may show all the stages of inflammation: the liquid infiltration, red, or dark red with retained tenacity of the lung tissue and a free flow of lymph when incised: the red hepatization with coagulation of the exudate, firmness to touch, a dry, granular aspect of the cut surface, and a marked friability under pressure; in the centre of each affected lobulette is seen the whitish engorged bronchiole with its infiltrated peribronchial tissue, the contents being early liquid and muco-purulent with excess of swollen granular epithelium; later it may be a firm coagulated mass, and later still a suppurating centre; gray hepatization may follow the red, but very commonly abscesses form in the centre of the lobulette, and, discharging into the adjacent bronchia, cavities are formed. Another

common result is gangrene, with softening and partial liquefaction. The gangrenous mass has a brownish, red or greenish gray color and a foul, putrid odor.

The smaller bronchia are blocked with exudate and infiltrated with leucocytes and fat globules, but the larger bronchia and trachea show a swollen, infiltrated mucosa, and contain a reddish froth, and on the surface a scum varying in color according to the nature of the material inhaled but usually of a greenish gray. Under this the membrane may show red patches of congestion, and erosions.

In cases implicating the pleura there is an extensive effusion of reddish or yellowish serum, and false membranes of varying thickness on the pulmonic and parietal pleura and even, it may be, on the pericardium as well. In this liquid are flocculi, granular matter, pus cells, and myriads of septic bacteria of divers forms. The process of decomposition often generates gas so that there is hydro-pneumothorax and an offensive smell. Generally diffused petechiæ betray the existence of septicæmia.

Symptoms. There may be the history of a predisposition, such as a hypostatic congestion from prolonged decubitus, or severe operation, with a full stomach, under an anæsthetic; or there may have been the manifest accident of inhalation of infected or silicious road dust, of irritant gases, smoke or hot air, of some irritant liquid given through the nose or forcibly by the mouth, or of solids in cases of impaired innervation of the throat, or even of blood in operations on the nose or throat. A history of coughing during drenching or swallowing is an important indication.

Another striking feature is the suddenness of the invasion and progress. Catarrhal bronchitis with fibrinous pneumonia usually sets in and advances gradually for days or even a week before it reaches its acme: inhalation broncho-pneumonia makes a sudden invasion and involves the smallest bronchia and the lung tissue in a few hours. This is favored by the solid or liquid form and the comparatively large size of the objects inhaled which are thus drawn with each inspiration deeper into the tubes, but also by the traumas effected by them on the mucosa and the active invasion of the wounds by the swarming micro-organisms.

The cough is hard, dry and deep, becoming later soft, loose and mucous, but always short and painful. The breathing is accelerated, often reaching 30 or 60 per minute and with perhaps a slight delay between inspiration and expiration, the pulse rises to 80 or 100, and the temperature to 104° to 106° F.

The difficulty of breathing rapidly encreases being accompanied by coarse mucous râles and a painful, rattling, suffocating cough. Early in the disease the expired air has a heavy, offensive odor and this goes on increasing to a marked foetor. The nasal discharge, at first serous, or mucous, becomes rapidly thick and purulent, having a yellow tint from escape of blood globules, and then brown from the necrotic products. It assumes a foetid odor at an early stage and becomes more and more offensive as putrefaction and gangrene progress. It may even show particles of the inhaled alimentary matters, coughed up, or debris of disintegrated lung tissue.

The case is marked by early and extreme prostration and debility, there is a complete loss of life and spirit, a growing stupor. drooping ears and eyelids, indisposition to move, and a generally hopeless appearance.

Percussion detects in both lungs, and especially in the lower third and anteriorly, limited areas of dullness,—representing the consolidated lobulettes, sometimes a lower flattened area terminating above at a horizontal line, and again zones of tympanitic resonance indicating emphysema or the formation of cavities.

On auscultation the bronchi in the upper part of the lung give coarse mucous râles, there may be crepitation around the consolidated patches, with mucous or sibilant râles, or heart or abdominal sounds transmitted through their centres. This may be modified at times by the amphoric sound, or, in advanced stages, by splashing or metallic tinkling from the hydro-pneumothorax.

In the last stages the dyspnœa becomes extreme, the animal is profoundly depressed by the toxin poisoning, he may be sunk in a stupor, and petechiæ on the visible mucosæ may betray the general septicæmia. A profuse and foetid diarrhœa may be among the latest phenomena.

Diagnosis. Apart from its history and the evidence of its causation, the disease may be at first confounded with bronchitis and especially the capillary form. But if it has supervened on

disease of the brain or digestive organs ; on tetanus or petechial fever ; on an operation done under anæsthesia or performed on the throat ; if there has been paresis of the throat ; if there has been drenching through the nose or forcibly through the mouth ; or if the affection has developed rapidly with high fever and much disturbance of breathing, prostration and stupor, inhalation should be suspected. If later there is foetid breath and expectoration, with a muco-purulent discharge, yellowish, brownish or chocolate colored, grumous or with particles of disintegrating lung or of inhaled food materials, and finally if there are objective symptoms of cavities in the lung, a conclusive diagnosis may be reached.

Prognosis. The result is usually fatal. Yet much depends on the lack of irritant or putrefactive tendency of the material inhaled and on the strength and vigor of the patient. We have had excellent recoveries from even a large amount of lard in the lungs, from inhalations of saline drenches and other medicinal agents, from blood and even small amounts of alimentary matters.

Prevention. With such a redoubtable disease the most important consideration is prevention. To this end all the causative factors mentioned should be guarded against. Drenching through the nose, a forcible administration of liquids through the mouth, especially in diseases that would impair innervation of the pharynx, the administration of liquids when the patient is down or with the head unduly extended, anæsthesia and operation with stomach full, prolonged decubitus on one side and bloody operations on the nose or throat without the precaution of tracheotomy and blocking of the windpipe above, should all be carefully guarded against.

Treatment. This is in the main that of capillary bronchitis or simple broncho-pneumonia, but special attention should be given to the removal of the foreign bodies or the arrest of their septic processes when possible. In case of blood in the bronchia the clots may be loosened, if not dissolved, by injecting soda bicarbonate solutions into the trachea with a hypodermic syringe or through an opening made in the windpipe.

To counteract putrefaction, antiseptic inhalations from tar, carbolic acid, creoline, cresyl, lysol, oil of turpentine, etc., are usually resorted to. Fumigations of dilute sulphurous acid or chlorine

may be tried. Peroxide of hydrogen (1 per cent solution) has a better effect, poured gently through the tracheotomy wound into the lung. This is a comparatively bland antiseptic, purifying for the time the air tubes and the foreign bodies, and tending to take up in suspension the debris of the latter and wash it out. The solution is poured in until efforts are made to expel it and it is in great part discharged carrying with it much of the offensive material. It may be repeated once or twice a day and need not interfere with the inhalation of gaseous antiseptics. Finally tallianine, 20-50cc, intravenously day by day may be resorted to.

INHALATION BRONCHO-PNEUMONIA IN CATTLE.

Causes. These are largely those already described for the horse. In administering medicine there is much greater danger than in the horse : *first*, because the agents are usually given in a liquid form, and *second* because when the nose is elevated the patient cannot resist swallowing liquids placed in his mouth and which pass at once beneath the very short, soft palate into the pharynx. In indiscreet drenching, therefore, when a constant current of liquid is flowing back from the mouth, some is inevitably drawn into the larynx in inspiration. Even water striking the laryngeal mucosa, rouses cough, and medicinal agents are more injurious in proportion to their irritant qualities. Once rouse a cough and it is followed instantly by a sudden and deep inspiration through the mouth which carries into the lungs any liquid left in the buccal cavity. The line of safety lies in not raising the nose too high, nor pouring the liquid in in a large or continuous stream, and in dropping the head instantly the moment there is shown a disposition to cough. Sacrifice any liquid that may be in the mouth (the loss may be estimated and added to the dose) and never run the risk of inhalation pneumonia.

A second practice which is even more reprehensible is that of dragging the tongue out and holding it fast during the giving of the drench. The movements of the tongue are essential to normal deglutition, and while it is true that forcible dragging on the tongue and straightening it, prevents the application of its root against the soft palate and the delay of deglutition even for an instant, it also assures a continuous flow of liquid into the pharynx, and some of this must pass into the larynx during inspiration.

Bouley adds the action of astringent draughts in constricting the fauces and pharynx and favoring the passage of food into the larynx. However this may be there can be no doubt that, as in the horse, contact of the pharyngeal mucosa and that of the margin of the larynx with local anæsthetics (ether, chloroform, chloral, carbolic acid, cocaine, etc.), lessens the alert control over the muscles and favors inhalation.

Oiled boluses which are said to slide more readily into the larynx (Albrecht), and the finely divided particles in gruels (Mazoux), and the acid element in cider (Cagny), are variously incriminated. Oils and melted lard, while less irritating to larynx and bronchia, are specially injurious in excluding oxygen and preventing æration of the blood, and elimination of carbon dioxide.

Inflammation of the pharynx and larynx—catarrhal, phlegmonous, or croupous—disturbs the sensibility and muscular control, and makes the giving of drenches much more dangerous, and the same has been noticed in pharyngeal tuberculosis and tetanus.

Tympany of the rumen is a great source of danger. The hurried breathing caused by the compression of the lungs by the diaphragm favors inhalation. The closure of the demicanal by the stretching of its pillars which spread out on the walls of the distended rumen, hindering alike the exit of gas from the viscus and the entrance of liquids descending the œsophagus, favors the return of the liquid to the pharynx by regurgitation, and its entrance into the larynx. Finally the compression of the œsophagus between the two pillars of the diaphragm, stretched by the forward pressure of the distended rumen, acts in the same way.

Milk-fever in cows (parturient paresis) has been in the past a common cause of inhalation pneumonia. In the general paralysis of the fully developed disease the pharynx participates, and when treated by drenches given by the mouth the descent of the medicament into the lung was a frequent occurrence. If the patient survived the coma, she had to face an attack of bronchopneumonia. Again from the arrest of digestive movements fermentation of the contents of the rumen with tympany frequently occurred, threatening regurgitation and inhalation, and in addition the prolonged decubitus on one side favored hypo-

static congestion of the lungs and paved the way for a severe attack when the alimentary or medicinal matters entered the bronchia.

Owing to narrow nostrils and less exposure at rapid paces on dusty roads, cattle suffer less than horses from inhaled particles of dust, yet when moved in droves the dust becomes a source of trouble. Other foreign bodies find their way into the lung by accident. Thus, in different cases of broncho-pneumonia, the following agents have been found in that organ: a bundle of hemp (Roch), some hog's bristles (Ujhelge, Leyendeker), a spike of rye (Strebel), masses of cotton in cattle fed uncleaned cotton-seed (Dralle), a broken fragment of a probang (Ujhelge).

The penetration of the lungs, pleura, and heart by pins, needles, and other sharp-pointed bodies, coming from the reticulum, should be mentioned as causes of pneumonia and pleurisy by foreign bodies, though these have not entered by inhalation, and the progress of the case is not from bronchia to lung and pleura, but rather from pleura to lung, pericardium and heart. Also penetrating wounds of the walls of the thorax, by which foreign bodies, (bullets, forks, spikes, nails, horns, tusks) have entered the lung and set up pneumonia.

Lesions. As occurring from materials inhaled, the broncho-pneumonia is most common in the lower parts of the anterior lobes, in which the lesions appear as multiple centers of inflammation with limits conterminous with the lobulettes, and with the interlobular tissue largely infiltrated. The affected lobulette stands out beyond the level of the adjacent lung, at first soft and infiltrated and later firm, hard, resistant, and friable, of a deep red, or dark blue color, with, in its center, the bronchia, filled with a fibrinous exudate, later becoming caseous, and containing the offending foreign body. The latter may vary in different cases noted above. As the disease advances gangrene appears, the lung becoming dirty white, brownish or greenish, and exhaling a foetid odor. The peribronchial connective tissue is infiltrated and often shows hard nodules which when caseated might be mistaken for tubercles. In other cases an extension is made to the pleura, causing effusions, false membranes, and gaseous products (pneumo-hydro-thorax). The bronchial glands are usually congested, swollen, indurated, and pigmented, and may show

multiple miliary abscesses, without the tubercle bacilli of the corresponding condition in tuberculosis. Miliary abscesses may even be disseminated through the affected lung. On microscopic examination the affected bronchioles and alveoli are seen to be filled with fibrinous material, red blood globules, leucocytes, and pus cells. The foreign body may be found lodged in this material, sometimes even in giant cells imbedded in it. The infiltrated portion may become the seat of abscess, or of necrosis.

In wounds through the walls of the chest the lesions are concentrated around the foreign body and its track, and vary with the structures involved. In foreign bodies traveling from the reticulum, the track of the body is usually in the form of a fistula, extending from the reticulum through the diaphragm, pleura, left lung and pericardium, having very thick infiltrated walls and it may be attended by effusion into the left pleural sac and pericardium. The foreign body is found at the anterior end of this fistula. In some cases the fistula does not extend to the pericardium, but toward some intercostal space.

Symptoms. The fit of coughing during the giving of a draught is usually the first symptom. It becomes increasingly difficult to give the draught without causing incessant coughing and the accumulation of froth around the lips. Mucous râles are heard in the lungs, the breathing is excited, and the animal blows its nose, shakes its head and moves its feet showing general uneasiness. These first symptoms may subside for a day or two, when signs of bronchitis and pneumonia appear. The respiration becomes increased, short, jerking, laborious, the nostrils dilated, the head extended and the fore legs placed apart with the elbows turned out. A loud mucous râle or clucking is heard in the trachea and bronchi, and a moan may attend on expiration. Percussion over the inferior and anterior part of the lung shows limited areas of flatness, transmitting on auscultation coarse mucous, sibilant, or blowing râles and ronchi, and each showing crepitation at its margin. As the disease advances an amphoric sound may develop implying the opening of an abscess and the formation of a cavity. In other cases the friction sounds may betoken the advent of pleurisy, or splashing or metallic tinkling the advent of hydro-pneumothorax.

In case of a foreign body passing from the reticulum, the

line of extensive exudation may sometimes be traceable by the dullness on percussion, or by a clucking sound on auscultation, and, as in pleurisy, there is usually a dropsical swelling, pitting on pressure beneath the lateral parts of the sternum or between the first ribs.

The constitutional symptoms, accelerated pulse, elevated temperature, suppressed secretions, are more or less marked in different cases. Fetid breath and expectoration are to be looked for.

Diagnosis. From lung plague it is distinguished by the presence of multiple small areas of dullness instead of one large area, by a more frequent and stronger cough, by the presence of foetid breath instead of the heavy mawkish odor of the exhalation, and usually by the more moderate fever and the absence of other cases in the herd, or of some evidence of infection. If there is a question of tuberculosis apply the tuberculin test.

Prevention has been fairly indicated under *causes*. The protection against inhalation of drenches consists mainly in only slightly elevating the head, leaving the tongue free, and pouring the liquid into the mouth in successive small quantities, each to be swallowed before more is given. Especially is this to be secured in drenching recumbent animals, and above all those suffering from milk fever, though the success of the modern treatment by udder distension virtually excludes the necessity of giving liquids by the mouth in that disease.

Treatment. In cattle, the issue is rather more hopeful than in horses, but the same general treatment is advisable. Beside the general treatment of the fever and local inflammation, the use of antiseptics (carbolic acid, creolin, lysol, chloro-naphtholeum) in volatile form, or even as liquid, is especially called for. As in the horse the best agent is solution of peroxide of hydrogen.

INHALATION BRONCHO-PNEUMONIA IN SHEEP.

Causes. This is a common trouble from the administration of liquid medicines, the causes being essentially the same as in cattle, and preëminently the holding the head too high, and pouring the liquid in a steady stream. Cases are on record in which an ear of wheat was found imbedded in a bronchium (Guillaume), and an ear of another graminaceous plant (Blanc).

Lesions consist in intense broncho-pneumonia, suggesting pulmonary œdema, followed by hepatization or abscess, which may burst into bronchium or pleura. Hence empyema is sometimes found with a variable amount of effusion and false membranes. Extensive gangrene of the lung may be present, and the generally diffused petechi of septicæmia.

Symptoms become rapidly developed, frequent paroxysms of coughing, with hurried breathing, fever, and a rapid emaciation. In milder forms the cough and loss of condition may be the principal symptoms.

Prevention should be sought, as *curative* treatment is usually fruitless.

INHALATION BRONCHO-PNEUMONIA IN DOGS.

Causes. The predisposition comes largely from inflammatory affections of the throat, as in distemper, which interferes with proper sensation and deglutition. The careless giving of drinks at such a time has been charged with half the deaths (Cagny), others are attributed to inhalation of dry sulphur (Albrecht), and others to dry jalap (Cadeac). It must be borne in mind that section of the vagus in dogs first showed the production of experimental broncho-pneumonia by the resulting nervous imperfection, and the interference with nervous control during agony operates in the same way. The congested and swollen tonsils, fauces and pharynx, covered with muco-purulent matter and even scabs and sloughs prove poor sentinels at the vestibule of the lungs, and more or less of the food materials or medicaments enter through the larynx. Worse than all, in ulcerous stomatitis the tenacious or inspissated exudate and sloughs of the gums and mucosa, swarming with bacteria, are liable to inhalation in the same way and form irritating foreign bodies of the worst kind, because of their dangerous microorganisms already accustomed to life in the tissues.

Other foreign bodies are inhaled by dogs which are breathing hard under violent exertion (dust of highways, or railway trains), or in hunting. Thus, Lechre found the thorn of a bramble in the centre of a pulmonary phlegmonous abscess which burst into the pleura with fatal results, and Weber, a spike of rye as the central factor in capillary bronchitis.

Lesions. These are found in the same order as in the horse. In very recent cases they may be mainly those of capillary bronchitis with the foreign bodies embedded in the exudate in the bronchioles. There may be *collapse of lobulettes* of lung connected with the inflamed and blocked bronchioles, such portions appearing depressed but red, consolidated and sinking in water. The affected bronchia are filled with a muco-purulent exudate containing many leucocytes, pus cells and detached and swollen epithelium. In cases of active septic fermentation, this exudate may be largely a thin incoagulable serum with more extensive infiltration of the coats of the bronchia, and of the peribronchial connective tissue. When the morbid process has extended to the alveoli and lung tissue, the air sacs are distended with the same serous fluid and the whole lobule is infiltrated so that it projects on the surface of the lung beyond other parts, and as numerous lobulettes are affected these stand out as bosses, of a red, brownish red, dark red, black, or grayish color. Still later the clusters of alveoli are liable to form purulent centres.

The exudate may be confined to the vicinity of the terminal bronchiole, forming a firm nodule without the implication of the entire lobulette until later. On section, this has a granular appearance due to the abundance of cell organisms,—red cells, leucocytes, pus cells, and swollen epithelial cells. This central nucleus is surrounded by a zone of splenization, of violet color and homogeneous structure but which is progressively invaded by the central granular gray nucleus. Multiple minute abscesses and vacuoles are found in advanced cases.

Œdema of the lung is not uncommon, and emphysema is also a frequent result of the violent paroxysms of coughing. Again the invasion of the pulmonary parenchyma may extend to the pleura, or an abscess may burst into the pleural sac causing a general infective pleuritis.

In all cases the exudates in the bronchioles, their walls, the air sacs and cells, the pulmonary tissue and the pleura, contain abundance of microbes of different kinds.

Symptoms. These are essentially those of capillary bronchitis advancing into pneumonia and even pleurisy. Disease may begin with a foreign body in a large bronchium, which will keep up a local inflammation for weeks or even months without extend-

ing to the bronchioles and lung tissue. When, however, these latter are invaded the symptoms are liable to prove distressing, as the disease advances by rapid strides. The breathing is hurried, and oppressed, sighing or panting, the patient sitting on his hind quarters, or standing, with his fore legs apart, his elbows turned out, the nostrils blocked by discharges, the mouth open, or if closed there are inflating of the cheeks and puffing of the lips, the projecting eyes and drawn muscles of the face express great anxiety, and the discharges are tenacious, filamentous, frothing or even bloody, and malodorous.

Percussion and auscultation reveal limited areas of flatness more particularly near the lower and anterior parts of the lung, surrounded by lines of crepitation, and in adjacent larger bronchia, mucous râles, which, like the heart and abdominal sounds, may be conveyed through the consolidated areas. The mucous râles may be combined with loud sibilant râles, or again, in the advanced stages, with amphoric sounds owing to the formation of cavities. Again all the successive symptoms of pleurisy may be present, notably, intercostal tenderness, friction sounds, later, flatness on percussion up to a given water line, the creaking sound of false membranes on the stretch, and metallic tinkling or splashing implying hydro-pneumothorax.

From the first there has been elevation of temperature and other indications of fever, which become more marked as the disease advances, and finally culminate in a profound prostration and debility, the result of the absorption of the ptomaines, toxins and waste matters generally. These elements, together with the defect in æration of the blood, and its being dammed back in the over distended systemic veins, lead to congestions elsewhere and particularly in the liver and bowels with serious disturbance of the digestive process. Vomiting occurs when even the blandest fluids are introduced into the stomach, indigestion, constipation and diarrhoea follow each other, and jaundice is not infrequent. In such a case the gastro-entero-hepatitis may be the cause of death.

Course. With inhalation of a large amount of alimentary matters or medicines, death may follow rapidly from blocking of the bronchia, which in the compact lung of the dog, break up quickly into the bronchioles. In other cases with less material inhaled or that which is less irritating or infecting, the progress

may be slow, advancing for several weeks before ending in death or recovery. In fatal cases the cough becomes weaker and ineffective; the respiration increasingly difficult, painful and exhausting, more and more rapid for a time but in the later stages tardy and even shallow—the respiratory centers having evidently become exhausted; the pulse becomes small and weak, almost imperceptible, though the heart may still beat tumultuously; the dog lies constantly on his sternum or side, careless of those about him, in a complete apathy or stupor and with the eyes deeply sunken. Finally death comes from the eighth to the fifteenth day as a result of asphyxia in a thoroughly poisoned system.

In favorable cases all the symptoms become gradually improved, the eye is brighter and fuller, the respiration easier and deeper, the pulse stronger and more regular, the cough less painful and more effective. Breathing is reestablished through the nose, and appetite returns. Relapses are, however, to be apprehended in connection with the rupture of abscesses, extensions from temporarily inactive centers, and even renewed inhalations.

Diagnosis will depend largely on the evidence of inhalation, on the progress of the disease from a capillary bronchitis to a pneumonia or even a pleurisy, on the rapid progress of the affection in its later stages, on the indications of the formation of abscess cavities, and on the fœtor and grumous character of the discharges.

Treatment. This is largely the same as for capillary bronchitis. More attention should, however, be paid to antiseptics. Careful attempts to wash out and purify the bronchia by the injection of hydrogen peroxide and the impregnation of the air with non-poisonous volatile antiseptics (carbolic acid, oil of turpentine, creolin, lysol, etc.) are especially indicated. Combined with warm water vapor, these are at once soothing, expectorant and antiseptic, and will often materially assist in clearing the invaded bronchia. As stimulant expectorants may be tried, carbonate of ammonia, alcohol or ammonium chloride vaporized by heat, or as a sedative agent, ipecacuan wine. The elimination of the toxic matters by kidneys or bowels must not be neglected. The fever and inflammation, must be otherwise treated on general principles, by antipyretics, derivatives, damp jacket and in the prostrate conditions, by diffusible stimulants and heart tonics. During convalescence a judicious combination of stimulants and biters, with pure air, sunshine and a generous diet will often be of material advantage.

CHRONIC FIBROUS PNEUMONIA. PULMONARY SCLEROSIS.

Synonyms. Interstitial Pneumonia. Cirrhosis of the lung. Induration of the lung. Pulmonary Fibrosis. Pulmonary Phthisis.

As usually seen this is rather a result of other inflammations and morbid processes than a disease by itself. It consists in a fibrous degeneration, extending from the bronchia, blood-vessels, interlobular connective tissue or pleura, and in which the exudate does not pass into active degenerative processes—granular, purulent, liquefactive, gangrenous—but becomes built up into new white fibrous tissue. It follows also on recovery after destructive changes of the lung with loss of substance as after abscess, or necrosis, with subsequent absorption and removal of the sequestrum, in the healing of vomicae, after miliary glanders, and in the seat of hydatids which have died and withered up. In such cases the cyst or the portion of tissue affected and removed, with more or less surrounding and organized exudate, forms a cicatrix, or, if larger, a fibrous or cirrhotic mass, which takes the place of the lung tissue. This is, therefore, in many cases rather a beneficial and reparatory process than a destructive one, nevertheless its existence impairs the capacity of the lung for respiration, and renders the animal permanently *short-winded*. It also predisposes to other lesions of the lung, such as emphysema, bronchiectasis, carnification, chronic bronchitis, etc.

SOLIPEDS.

Causes. Chronic fibrous pneumonia may result from the acute fibrinous form, from broncho-pneumonia, from contagious pneumonia, from influenza, from capillary bronchitis and, above all, from chronic pleurisy and hydrothorax. Morbid deposits like glanders, actinomycosis, so-called botriomycosis, aspergillus, and ecchinococcus may also be incriminated. A condition favorable to cirrhosis is an exudation in small amount of a strongly nucleated lymph, which being near a sufficient blood supply, and having ample plastic cells present, tends at once to organization. Hence it is especially liable to follow on chronic bronchitis, chronic pleurisy, hydrothorax and even the irritation

caused by foreign bodies which tend to become encysted. In chronic bronchitis, which is so common in solipeds, it extends in a branching form along with the air tubes, involving in different cases bronchiectasis, constriction of bronchia, emphysema, compression and obliteration of air sacs and alveoli, collapse of the dependent lung tissue, hence limited to lobules, which become fibroid, compressed and airless. When the fibroid condition forms around an encysted foreign body it is primarily circumscribed, but as the offending object is usually located in a bronchium, the blocking of its lumen is entailed and the condensation and fibroid degeneration of the dependent lobule or lobules follow. When the process starts at the pleura, its extension is mainly at first in the interlobular connective tissue, and the lobules are first compressed, deprived of air and then invaded by the fibroid development. Here the main difference is the comparative immunity of the peribronchial tissue, the compression and fibrous formation having advanced from the pleura and interlobular tissue instead of from the branching air passages. In cases of sclerosis from miliary glander deposits, the fibrous framework of the lung is more universally involved without indication of the bronchia or pleura having been primarily or specially invaded.

Lesions. Beside the general encrease of fibrous tissue, which has contracted around, compressed, and caused atrophy the enclosed lung, there is a remarkable firmness and cohesion of the tissue, so that it is easily distinguished from simple hepatization. It may be red or gray, and like the hepatized lung is devoid of air, and sinks in water, but it is relatively heavier and sinks faster and deeper. In the earlier stage of red consolidation there is little blood escapes on section, and microscopically the alveoli appear filled with a yellowish exudate. Later, with the grayish condensation these are usually empty and with their walls in contact. Where the primary lesion has been a broncho-pneumonia the peribronchial fibroid condensation is especially marked, the bronchia, dilated or constricted, may contain a soft caseous debris or pus collection. Outside the peribronchial thickening, the lung tissue may be represented for some distance by dense fibrous septa and pockets of pus some of which communicate with each other and some with the bronchia. Even where sup-

puration has not ensued, the affected parts of the lung, with the lung tissue compressed and atrophied, have assumed a grayish hue. In cases that have followed pleurisy the lower portions of the lung are especially affected, but the whole affected lobe may have a dense fibrous envelope (the condensed false membrane), from which septa pass into the lung, circumscribing the lobules and determining parenchymatous softening and absorption. In such cases the bronchia are usually unaffected.

The fibrous structure may become as hard as cartilage and grate under the knife when cut. The sclerosis may appear at any point, and no matter what may have been the point from which the disease started.

The right heart is usually dilated by the blood pressure caused by the obstruction to circulation through the lungs, and the tricuspid valves become insufficient.

Symptoms. These are very varied and not always diagnostic. In case of a fibrinous or broncho-pneumonia or a pleurisy, it may be suspected when the fever subsides without clearing up of the lung, when, on the contrary, hurried breathing and cough continue unabated, and are easily roused or greatly aggravated by even moderate exertion. Perspiration is easily induced and the patient is unfit for work. The chest remains expanded and the nostrils dilated. Most characteristic of all, the consolidated parts of the lungs do not become more resonant to percussion, nor does the respiratory murmur return in them.

For a time the appetite and spirits may be good, yet the patient loses flesh, and both pulse and respiration are easily accelerated, and accompanied by a muco-purulent discharge. Later appetite fails, breathing is made with double lift in expiration, cough becomes paroxysmal, and emaciation advances rapidly.

If it supervenes on broncho-pneumonia, the sibilant and mucous râles are prominent in the affected parts, a cavernous blowing may be present, the painful cough occurs in fits, and the muco-purulent discharge is often foetid. New attacks of bronchitis are liable to occur at intervals each leaving the subject worse than before, and under these conditions the cough becomes more frequent and painful, dyspnoea more trying and the double lift of the flank more marked.

When it advances from pleurisy, there is the gradual weakening of the respiratory murmur over the affected area, and a slow decrease of resonance on percussion.

Diagnosis. The most diagnostic signs are : the preliminary evidence of inflammation in the chest, followed by a marked decrease of temperature, perhaps to normal, improvement of pulse, and appetite, without any lessening of the percussion flatness, or any increase of the area of respiratory murmur. The persistent breathlessness, weakness and perspiration under exertion are also marked features.

From tumors it is distinguished by being less circumscribed, by the cough and the discharge.

From hydrothorax, by the absence of the dullness on percussion up to a given horizontal line only.

From emphysema, by the marked discharge under exertion and the absence of the extensive drum-like resonance, on percussion, over a large area of lung, and by the soft, mucous character of the cough, at intervals if not continuously, in contrast with the uniformly dry, weak, husky and abortive cough or heaves.

Treatment must be essentially preventive as little can be expected of a lung in which firmly organized fibrous tissue has taken the place of the pulmonary tissue. Every effort should be made to favor removal of exudates in inflammation, and to prevent their organization. This comes under the general headings of capillary bronchitis, pneumonia and pleurisy. But even when all acute inflammatory action has ceased, and the exudates have been partially organized, some reabsorption may be looked for in favorable cases. Stimulant expectorants (oil of turpentine, terpine, terpinol, balsam of Tolu or Peru, tar water, guaiac, chloride or carbonate of ammonia) may give good service. Alkaline diuretics (bicarbonate of soda, nitrate or acetate of potash, iodide of potassium) will serve to liquefy and favor removal. Diuretics greatly favor absorption. Tonics, especially those with a special action on the nerves of the respiratory system and nutrition, (arsenious acid, arseniate of soda, arseniate of strychnia) are not to be omitted. Finally nourishing food, good hygiene and even breathing exercises are of great service. As massage disperses the exudations in surface parts through adjacent lymph plexuses and favors absorption, so the respiratory movements, in a lung no

longer inflamed, favor movement and absorption of exuded products. Moderate, regular and gradually increasing exercises to increase the breathing becomes therefore a valuable aid in cases of this kind. If improvement is not shown in one month of treatment the case is usually hopeless, and if unfit for work or breeding the patient may well be destroyed, to avoid further outlay in keeping it.

CATTLE.

Causes. The bovine races are more subject, than the horse to fibrous degeneration of the lung, mainly because there is less tendency to suppuration, and also because the excess of connective tissue in the organ makes a very favorable basis for the formation of fibrous tissue. It may succeed almost every form of pulmonary inflammation (fibrinous pneumonia, broncho pneumonia, inhalation pneumonia, tuberculosis, lung plague) and parasites (strongylus micrurus, S. pulmonaris, echinococcus, cysticercus tenuicollis, distoma magnum, aspergillus, actinomyces).

Lesions. The morbid anatomy is, in the main, as in the horse, and like that varies according to the original cause. In lung plague, or the contagious pneumonia of cattle, a large extent of lung may be involved at one place, or if following a sequestrum it may be confined to narrower limits—a firm, small cicatrix: if from broncho-pneumonia there will be evidence of its having extended in a branching manner with the divisions of the bronchia; if from pleurisy the fibrous plates will be most marked between the outer lobules, while the bronchia will be largely exempt; in hydatids there will be the small circumscribed nodules of sclerosis. If from pleurisy there are usually extensive adhesions, and often implication of the pericardium. In all cases in which advanced and extensive fibroid degeneration exists, the obstruction to the pulmonary circulation has determined more or less dilatation of the right heart, and in many cases insufficiency of the auriculo-ventricular valves. In advanced cases also emphysema is a marked feature of the, as yet, pervious portions of the lung.

Symptoms. Failure to establish an early convalescence after an acute inflammation of the air passages; a persistent cough, dry, rarely moist, roused by driving, or pinching the back; hurried or even difficult breathing under exercise; wheezing or sib-

ilant râles in the chest ; a reduction of the percussion resonance over given areas, with increased or even drum-like resonance around it ; dry, withered hair, sometimes erect on chine or back ; hidebound ; dry muzzle ; drooping ears ; slight tympanies ; constipation or diarrhœa.

The idea of tuberculosis should be excluded by a tuberculin test without reaction, and by the absence of indications of tubercle elsewhere. Disease of the heart should be eliminated by a careful test of its sounds, rhythm and area.

Treatment. In the early stages treatment would be as in the horse. With advanced conditions of disease it would be fruitless. A good judgment may be formed on the recent nature of the lesions, the usefulness of the animal for milk or breeding and the ease with which she might be fattened for the butcher.

DOG.

Causes. As in other animals *chronic fibrous pneumonia* is liable to supervene on an acute attack : (distemper, capillary bronchitis, broncho-pneumonia, pneumokoniosis or dust-inhalation pneumonia ;) as in cattle it is very liable to result from tuberculosis, and again it follows parasitic invasion (*strongylus vasorum* or *subulatus*, *filaria*, *uncinaria*, *spiroptera*, *aspergillus*.)

As in the other animals it is a lighter form of inflammation the exudates of which can form into new tissue. Old dogs that have suffered from chronic bronchitis or inhalation pneumonia are especially liable to suffer in this way. Tuberculosis is a not uncommon condition, cicatrices of healed cavities, and extensions into the lung being met with. Inhalation of coal dust or soot has again and again been incriminated and the anthracnosis has even been produced by keeping dogs in a room lighted with ill-burning oil lamps. The fine particles of carbon which adhere to the bronchial mucosa are mostly carried out by ciliary action and are coughed up. That which reaches the alveoli being beyond the reach of the cilia, becomes englobed in the wandering leucocytes and reaching the lymph plexuses in the pulmonary connective tissue, is in part deposited mapping out these spaces as dark lines : a part proceeds along the lymph vessels in the lymph glands which filter out enough to give them a dark pigmentation ; finally, a small amount reaches the thoracic duct, and

veins and is finally deposited in the liver and spleen. In the case of the *strongylus vasorum* the sclerosis is primarily concentrated around the bronchia and larger pulmonary blood vessels. It is no uncommon thing to find around the infested bronchia, nodules consisting mainly of embryonal cells, in the midst of which are the ova and embryos of the strongle. When the strongles have passed the larval stage and migrated into the vessels, these nodules remain as fibroid masses, and the degeneration may extend into the adjacent lung.

Lesions. These are largely like those of solipeds. The sites and extensions of the fibrous formations show the nature of the primary lesions, and in the case of anthracnosis the pigmentation of the lung and especially of the connective tissue is significant. This pigmented condition must not, however, be inferred from the small amount of black pigment found on the surface of the healthy lung in the dog.

Symptoms. These vary according to the grade and extent of the fibroid degeneration. In advanced cases, though the temperature is normal, the difficulty of breathing is extreme or is roused by the slightest exertion. This is accompanied by an irritating cough. Wheezing and sibilant sounds or blowing râles are heard in the lungs, and percussion shows a loss of resonance (even complete flatness) over given areas. There is increasing emaciation and the dog is practically useless. Imperfect cardiac valves may be detected.

Treatment. This would be only hopeful in the line of prophylaxis, obviating the sclerosis by successful treatment of the earlier and causative lesions. This would be undertaken along the same general lines as in the horse. In advanced conditions it becomes at once merciful and economical to do away with the animal.

ACUTE SERO-FIBRINOUS PLEURISY IN THE HORSE. PLEURITIS.

Causes, cold, damp, soils and exposures, as with rheumatism, youth, vigor, heavy diet, digestive, or hepatic disorder, over exertion, perspiration and succeeding chill, wading or swimming rivers, standing in snow, salted snow, rain, sleet, snow, draughts between open doors and windows, clipping, cold sponging of legs, tuberculosis a common cause in man and cattle is rare in horses, surface pneumonia, cancers, actinomycosis, tumors. Traumas from broken rib, penetrating intercostal wound, blows, contusions, ruptured pulmonary or intercostal abscess. Irritant (infectious) exudate suggests microbes. Symptoms, chill, reaction, partial sweats, pawing, pointing one foot, hyperthermia, hard, jarring pulse, hurried breathing, inspiration catching, pleuritic ridge, uneasy movements, hacking cough, tumors and twitching of chest muscles, tender intercostals, grunting, friction sound, subsiding with appearance of dull area below, signs of effusion, relief, dyspnoea, lifting flanks and loins, perspirations, stocking limbs, pasty swelling on sternum, effusion, of same level on both sides, creaking sounds, splashing, gurgling, metallic tinkling, weakness, sinking. Signs of adhesions, compression of lung, abscess. Duration. Lesions, early formation of false membranes, pleuritic effusion, its composition, its color at different stages, dry pleurisy, sero-fibrinous, sero-fibro-purulent, hydro-pneumothorax, tubercle. Prognosis. Treatment, during the chill, warm air, clothing, drinks, injections, compresses, pilocarpin during early inflammatory stage, derivatives, dry cupping, mustard, cantharides, hot water, or air, cold applications, laxatives, calmatives, anti-rheumatics, alkaline agents, with bitters, diuretics, heart tonic, iodine, mercury, thoracentesis.

Definition. This is an acute or chronic inflammation of the pleura with a sero-fibrinous exudation into its cavity.

Synonyms. Pleuritis, Morbus pleuriticus, Morbus lateralis, Pneumonia pleuritis. Peripneumonia. *Fr.*, Pleurésie. *Ger.*, Scitenstich, Brustfellenzündung. *It.*, Pleurisia. *Sp.*, Pleuresia.

Causes. Pleurisy is common in all domestic animals and especially so in cold, damp, exposed localities which suffer equally from rheumatism. It occasionally extends to the fascia of the limbs, the joints, or the navicular or other trochlea as a rheumatic affection. The disease is prevalent among young and vigorous horses, four or five years old, on stimulating feeding. Here hepatic derangements and poisons, over-exertion, perspiration and succeeding chills are especially to be expected. Plunging the limbs in ice cold water as in wading a river (Fromage), standing in snow and, above all, in salted snow, or facing a cold

rain, sleet, or snow when perspiring or fatigued, are recognized causes. A full drink of ice cold water when freely perspiring, and followed by standing in the frosty air, or in a cold current indoors. Exposure unblanketed after clipping in winter (Field, Trasbot), and even sponging the body or legs with cold water when heated, or fatigued, or both. St. Cyr found that pneumonias stood to pleurisy as 3:1, Trasbot as 10:1, yet the latter draws attention to the fact that in cavalry horses habituated to the stable and sent out into camps in the depth of winter, the pleurisies are more numerous than pneumonias. This may suffice to show the importance of the rôle filled by cold and chill in the production of pleurisy. Yet many physicians look upon the chill as a predisposition only, while the true origin of disease is microbian. And in man a large proportion of pleurisies appear to be distinctly tuberculous. Bowditch traced 99 cases of acute pleurisy and found that 32 afterward proved tuberculous. In Germany 13 out of 15 pleuritic persons reacted under tuberculin. City water injected produced pleurisy (Leblanc and Trousseau). The objection to generalizing too largely on this for the lower animals is that the horse and dog, in which tuberculosis is rare, are by far the most common subjects of pleurisy, whilst cows which are very prone to tuberculosis show few cases of simple pleurisy. Again, we find pleurisy in the horse as the result of other diseases localized in or adjacent to the pleura, and where there is nothing to indicate tuberculosis. Thus it follows pneumonia approaching the surface of the lung, cancers, actinomycosis and other tumors, and traumas—a pulmonary abscess bursting into the pleura, a broken rib scratching and lacerating the lung, a perforating wound of the intercostal space, or in cattle a sharp pointed body advancing from the reticulum toward the heart.

But the presumptive absence of the tubercle bacillus in the great majority of pleurisies in the horse does not prove the absence of all pathogenic microbes. Trasbot, who rejects the microbian theory, found that the injection of a little of the exudate into the pleural cavity of a sound horse, always determined a generalized pleurisy. Injections of distilled water or the fluid of hydrothorax with the same antiseptic precautions, made separately by himself and Laborde and by Cadeac had no pathogenic effect. Trasbot attributes the pleurisy vaguely to the irritant effect of the exudate,

but it is clear that pleurisies are usually complicated by microbial invasion even if this should be secondary rather than primary. There are forms of pleurisy which are primarily the result of microbes, as in lung plague, contagious pneumonia, influenza, canine distemper, glanders, tuberculosis, pneumo-enteritis, actinomycosis and theroretically it might be supposed that in our ordinary acute pleurisies, other germs that have been lurking harmless in the system may take occasion, by reason of the lowered vitality induced by a chill, or a trauma or fatigue, to colonize the thoracic serosa and develop pleurisy. Under such a theory, the predisposing and microbial factors would remain equally effectual, but only operative when conjoined, neither being strongly pathogenic without the other. Still in inhalation broncho-pneumonia the pleura clearly suffers through the microbial invasion from the lungs.

Until the constancy of the microbial factor is demonstrated we must recognize the time-honored doctrine, that pleurisy may be due to cold, exposure, over-exertion, to traumatic injuries, blows, concussions, fractures, penetrating wounds and to extension by contiguity from adjacent diseases. Yet microbes rarely fail to invade the inflamed pleura.

Most commonly pleurisy is unilateral on the right side but is often on the left or on both sides.

Symptoms. There is the early symptom of shivering followed by a hot stage in which the limbs participate and partial sweats bedew the surface. There are first uneasy movements of the fore limbs with some lifting of the flanks and this discomfort increases until the patient is panting with pain and occasionally glancing round at his heaving flanks and even pawing as in colic. He may even lie down carefully but quickly gets up again. If the pleurisy is confined to one side the corresponding fore limb is often advanced before the other. The nose may be turned at intervals toward the affected side. The temperature is 102° and upward. The pulse is quick, hard and incompressible, being usually compared to a jarred wire and beats from 48 to over 60 per minute. The breathing is highly characteristic. It is hurried, is carried on chiefly by the abdominal muscles to avoid the rubbing of the inflamed pleuræ on each other, and has the inspiration short and suddenly checked by an audible closure of the glottis while the

expiration is slow and prolonged. This character of the breathing is well observed when the ear is placed against the false nostril. The ribs are drawn out and fixed. The laboring abdominal muscles stand out as a ridge from the outer angles of the ilium along the lower ends of the last ribs (pleuritic ridge). A tremor on this line is often noticeable in the early stages. It may also be felt by the hand laid on the costal region. The horse does not stand obstinately still as in pneumonia, but frequently moves as if seeking an easier posture. When moved or turned he will groan at each step, much more so than in pneumonia, and he moves unsteadily and uncertainly, as from nervous or muscular weakness. The loins are insensible to pinching. Fæces are small, dry, hard balls, few in number; urine is devoid of chlorides, scanty, dense, and high colored; appetite gone; thirst craving. Yet a swallow of cold water may cause pain and even a moan (Barreau). The mouth is dry and clammy; The eyes dull and may be semiclosed. The short, hacking cough contrasts with the deep, rare cough of pneumonia. The expired air is not so hot, nor the mucous membrane of the nose so red as in the last named disease and there is no nasal discharge. A twitching of the muscles of the chest is sometimes seen and if the intercostal muscles are pressed upon, the animal winces and frequently grunts. The last symptom is likewise seen in rheumatic disease of the intercostal muscles (pleurodynia) but the absence of the fever, the cough, and other chest symptoms sufficiently distinguishes this. Auscultation detects in the early stages, in addition to a healthy respiratory murmur, a friction sound, audible in inspiration only, in short jerks near the close of the act and comparable to the rubbing of the palm of one hand over the other laid over the ear, but this is no longer heard when effusion of liquid has taken place into the pleuræ. Percussion in the early stages detects no change from the healthy chest resonance.

If not relieved in from twenty-four to thirty-six hours, a remarkable modification of the symptoms takes place indicating the occurrence of effusion. The violent symptoms are suddenly abated. The quick catching breathing which is in many cases accompanied by a grunt, becomes easy and though fuller than natural is comparatively regular. In particular the inspiration is free and full and comparatively painless, the sudden check and

the grunt by which it was arrested having alike disappeared. The tension of the abdominal muscles and the tucked up appearance of the flanks give way ; the pulse acquires a softer character, the haggard, pinched countenance is relaxed, and a general appearance of comfort and even liveliness prevades the animal. This temporary improvement is often so great that the horse will take to feeding as if he had all at once recovered.

The apparent recovery is, however, only transient. Soon the pulse becomes more frequent and loses its fullness, the breathing is more laborious and attended with a characteristic lifting of the flanks and loins, the nostrils are widely dilated, the limbs outstretched and the elbows out-turned, the eyes stare and project and the countenance has a haggard appearance indicating threatened suffocation. Partial sweats may break out on the surface, due to the state of nervous excitement and general relaxation and supplementing in some degree the impaired exhalation from the lungs. Auscultation over the lower region of the chest shows a complete absence of the respiratory murmur, rising to the same level precisely at all points. Percussion elicits no resonance on the same region. If the effusion has taken place slowly or existed for some time, the dullness and absence of sound will usually indicate that the liquid rises to the same level on both sides. So thin and permeable is the posterior mediastinum in its lower part that unless thickly coated by new solid exudations, the effusion readily passes through it and rises to the same height on both sides. If gas as well as liquid is produced in the pleural sac a gurgling or splashing sound may be heard on auscultation, and occasionally, after rising or other change of position, a *metallic tinkling*, due to droppings from the shreds of the false membranes above into the fluid below.

As the disease proceeds dropsical effusions are observed beneath the skin of the breast and abdomen and into the limbs, a mucous rattle is heard in the trachea, a clucking sound may be at times heard over the chest, the nose, ears and limbs become cold, the pulse increases in rapidity and weakness, shows the distinct anæmic tremor or thrill, and becomes rapidly imperceptible ; the horse moves unsteadily and often falls suddenly dead.

This early fatality is, however, only seen in the worse cases. In those about to terminate favorably, improvement is shown

usually about the fourth day. The lifting of the flanks and loins becomes moderated, the ribs move more freely, the grunt ceases, the pulse is fuller, softer and less frequent, and auscultation and percussion show a steady decrease in the effusion. The loins become sensitive to pinching, and chlorides appear in the urine. Appetite meanwhile returns, the horse moves more freely, lies down for a length of time in succession, and convalescence lasts from two to three weeks.

In the less fortunate cases structural changes more or less permanent, keep up symptoms of illness for a variable length of time. Sometimes after the liquid effusion has been absorbed the lung remains attached to the side of the chest by newly formed tissue (false membrane) and while this is undergoing a drying and organizing process, it gives rise to a leathery, creaking sound, heard on auscultation and easily mistaken for crepitation. Sometimes an abscess forms on the surface of the pleura or in the newly organized false membrane, and either bursts into the pleural sac (empyema) where it serves to infect, and increase and sustain the irritation, or it makes its way through the intercostal spaces and is discharged externally. In this last case its advance toward the surface is heralded by an extensive inflammatory infiltration and pasty swelling much more tender to the touch than the dropsical swelling already referred to. Another condition is that in which false membranes of considerable thickness invest a lung and, following the law of all fibrous structures in process of organization, they contract and cause a compression and partial collapse of the contained lung tissue. A flattening of the corresponding side of the chest and a muffled and almost inaudible respiratory murmur is the result of this condition. In some measure these symptoms are present during convalescence in all cases of pleurisy since the lung never expands to its full size till some time after apparent recovery, but it is only when the organ is invested with false membrane that the symptoms are very apparent.

In all such cases of prolonged pleurisy from protracted structural change, there is continued illness without the violent symptoms by which the acute form of the disease is manifested. The acute suffering, the restlessness, the grunt, and even the catching breathing may be absent; the temperature may be almost reduced to the healthy standard, the pulse small and tolerably soft, the

appetite considerably improved and the different secretions nearly normal; yet the pinching of the intercostal spaces causes sharp pain, and measurement, auscultation and percussion testify to the persistence of disease. The animal is hidebound, unthrifty and unequal to any exertion. The cough is weak and painful and sometimes accompanied by a grunt.

Beside the changes connected with exudation and effusion, and organization or suppuration in the exuded products, *gangrene* sometimes results. A case of this kind is related by *Percivall*.

The *duration* of pleurisy may thus extend from two days in very acute cases to several weeks, or even months if we estimate it by the continuance of *hydro-thorax* in the chronic cases.

Post Mortem Appearances. These consist mainly in the presence of false membranes, lining the pleura and hanging in cobweb-like shreds into the cavity of the chest, and of the liquid effusion which fills up the chest at its most dependent part. The pericardium also contains fluid in many cases. The successive changes are as follows: 1st. Capillary dilatation causing arborescent congestion of the pleura, with punctate redness at intervals ineffaceable by pressure; 2d. Swelling, proliferation and disquamation of the epithelium, roughening and drying the surface; 3d, and sometimes, in 24 hours, exudate into the serosa, with formation of embryonal cells, a sero-fibrinous effusion into the pleural cavity, and the coagulation of the fibrine, first on the inflamed surface as false membrane; 4th, The false membranes are encreased layer upon layer, may become torn or shreddy, cause morbid adhesions, or compress and condense the lung. They become vascular and form white fibrous tissue, from within outward by a process comparable to granulation in an ordinary sore. The periods at which exudation takes place, and when the principal changes take place in the exuded materials have been well investigated by Dupuy, Hamont, Delafond and St. Cyr. They induced pleurisy by injecting irritant liquids into the chest, and noted the regular sequence of changes.

Dupuy injected two drachms of oxalic acid dissolved in three ounces of water. Symptoms of pleurisy at once came on, with the friction sound characteristic of its early stages. Next day friction sound had ceased and evidence of effusion existed. The same experiment repeated on several horses showed that if killed

at any period subsequently to this, considerable exudation had already taken place. In one horse in which the disease was of 50 hours' standing, the chest contained 43 pints of citrine-colored fluid, and abundance of yellow, thick, false membrane enveloping the costal and pulmonary pleuræ.

Hamont injected seven ounces of a weak solution of tartaric acid into the left pleural sac, repeated the injection next morning and destroyed the horse twenty minutes afterward. The chest opened immediately showed a small amount of liquid on the affected side, and the pleura injected and reddened.

Delafond made twenty-two experiments with the same general result.

Percivall found recent adhesions between the lungs and side as early as seventeen hours after the commencement of the pleurisy.

Andral injected rabbits with acetic acid and in nineteen hours found in the injected pleura soft, thin, false membranes, traversed by red anastomosing lines, and in certain cases a serous or puriform fluid.

W. Williams found a false membrane formed, twenty-four hours after the injection of the irritant.

St. Cyr, in a series of 43 experimental and casual pleurisies in horses, found that in a very few hours there were marked local congestion and swelling of the pleura speedily followed by the formation of soft, pulpy, friable false membranes, largely amorphous and granular but impregnated with many cells and nuclei. These adhered feebly to the pleura but sometimes accumulated with prodigious rapidity so as to cover in three or four days the whole pleural surface on one or both sides. The attendant serous effusion was bloody, turbid, or lactescent. The pleural surface under the false membrane was highly vascular and studded with fragile, red, conical elevations projecting into the membrane. Exceptionally the sub-serous connective tissue became the seat of exudation as well.

From the sixth to the ninth day the false membranes began to become vascular and from the tenth to the fourteenth day commenced to organize into connective tissue. With the advent of this stage, the inflammatory action tended to subside, and the reabsorption and repair to ensue.

Pleuritic effusion. This varies greatly at the different stages

of the disease. As effused it has a composition resembling that of the blood :—

Water,	-----911 to 924
Albumen,	-----63.33 to 82.50
Fibrine formers,	-----2.16 to 12.50
Extractive matter.	
Salts.	

The progressive changes from the hæmorrhagic effusion to the limpid hydrothorax and their relation to the different stages of the disease and the subsidence of the inflammation are of the greatest importance in deciding questions of responsibility, when the animal has recently changed hands. St. Cyr has classified his cases in the following instructive table :

Duration of the Disease.		Effusions.				Total.
		Port Wine	Sero-sanguineous.	Muddy or Grayish.	Limpid.	
From	1st to 7th day.	9	6	3		18
"	8th to 15th day.	2	3	4	6	15
"	16th to 30th day.		1	1	5	7
After	30th day-----				3	3
		11	10	8	14	43

Up to the 7th day 50 per cent were dark red ; after the 7th day only 13.3 per cent ; and after the 15th day none. Up to the 7th day 83.3 per cent were either dark red or sero-sanguineous and not one had attained to translucency. After the 7th day only 8 per cent were of port wine hue, and by the 15th day 25 per cent of all cases of over seven days standing were already transparent. Of all cases of over 15 days standing, 80 per cent were perfectly translucent and none showed the dark red hue. Finally, after the 30th day all remaining cases were limpid. This of course must not be applied with the same confidence in both directions. While translucency of the effusion bespeaks seven days' standing and probably fifteen or twenty, the dark red hue must not be held to imply a recent date for the attack. A relapse in the course of convalescence may easily and quickly stain anew a liquid that was already limpid, or had advanced far toward this condition.

The appearance of the lung tissue in a case of confirmed pleu-

risy is characteristic. The lung is of a dull red color, shrunk, slightly collapsed, flabby, scarcely crepitant under pressure and heavier than water or floating in water. It is tough, not friable like hepatized lung, and its cut surface is dry, smooth, and presents the interlobular septa very well marked. This is due to the compression by effused liquid, and by the organizing and contracting false membranes covering the lung, and implies nothing more than simple condensation. The air cell may be collapsed, but contains no new product and has not parted with its epithelium, and the lung can be inflated through the bronchia.

Differentiation according to the nature of the effusion. Pathologists have divided acute pleurisy into the *dry*, *sero-fibrinous*, and *sero-fibro-purulent*.

1. **Dry or fibrinous pleurisy** has usually the most acute type and the exudate containing an excess of the fibrinogenous elements forms a coagulum or false membrane on the affected surface, tending to bind that to the part adjacent—the lungs to the costal pleura. The serum, small in quantity, is in the main retained in the exudate or if set free is actively absorbed by the healthy pleura,

2. **Sero-fibrinous pleurisy.** This form is usually less acute and more extended, involving perhaps an entire pleural sac, or even both sides of the chest. This is the common form of pleurisy and is that referred to in the experiments of St. Cyr and others above. The earliest lesions in experimental cases (with chloride of zinc solution) in dogs are an uniform bright red congestion, with a bright, shining surface as yet perfectly dry. There is already shedding of patches of the endothelial cells, swelling and proliferation of the superficial connective tissue cells, and the formation of a few pus globules. This is seen in from half an hour to six hours after the application of the irritant.

Next follows the exudation of fibrine and serum, which respectively coagulates as false membrane on the inflamed membrane, or drops to the bottom of the sac as liquid. The fibrine appears as granules, little knobs and threads between and on the endothelial cells and entangling a few pus cells. The changes are now much more marked in the connective tissue cells, which are more numerous, larger, nucleated and often stellate or polygonal. Changes are well advanced in twenty-four hours. The cells go

on increasing to the fourth or fifth day, when new blood vessels are formed into the membrane and may be injected from the pleura. From this time, in favorable cases absorption of the liquid proceeds, and the fibrine is organized, and by the fourteenth day is transformed into connective tissue, the superficial cells forming endothelium, and the deeper, branching connective tissue cells. The result is the thickening of the pleura and the formation of adhesions. The case, however, may prove fatal, or it may be protracted through the continued production of fibrine and serum, or it may pass into empyema.

3. **Sero-fibrino-purulent pleurisy. Empyema.** This is usually very dangerous as well as complicated. It may supervene on the last described form. It may depend on rupture into the pleura of abscess of the lung, bronchial glands, liver, diaphragm or intercostal space, and the infection of the chest cavity. It may in the same way follow the laceration of a bronchium by a broken rib, the perforation of the intercostal space by a foreign body, or (in cattle) the penetration of the chest by a sharp-pointed body from the reticulum. It may follow at once on pleurisy of a very high grade. Probably in all such cases there is infection of the pleura by pus microbes. When there is a communication with a bronchium, the reticulum or the external air, there are usually septic germs in addition, and the contents of the chest become foetid.

The purulent fluid may accumulate in the lower part of the pleural sac, or it may be confined in abscess form in the false membrane, and extend thence into surrounding tissues. The pus-containing pleura, or cavity infected by the pus germs, assumes the appearance of a granulating surface or of the lining membrane of an abscess, and continues to produce pus in greater or less amount.

The formation of pus in the pleura is known as *empyema*. When air enters the pleura through a wound perforating the chest wall, or when gas is formed in the pleura, the condition is **pneumothorax**. As liquid is usually present as well, it is **hydro-pneumothorax**.

Tubercular and other forms of pleurisy have in certain cases been superadded to the specific local lesions, by which such diseases are individually characterized.

Prognosis. Occurring in an otherwise healthy system and especially if confined to one side of the chest, pleurisy is not frequently fatal, and under appropriate treatment recovery is oftentimes rapid and satisfactory. A certain number of cases merge into chronic hydrothorax, the inflammation apparently subsiding, but absorption failing to take place. The hydrothorax may last for months or even a year.

Treatment. If seen during the *chill* and before inflammation has been definitely established, every effort must be directed to secure its abortion if possible. No time should be lost in placing the patient in a warm, comfortable stall or box, covering him with woolen blankets, and actively rubbing and loosely flannel bandaging the legs. Warm drinks and warm injections must be given. Half an ounce or an ounce of camomile or boneset in infusion, in two or three quarts of hot water, or in the absence of this, any of the carminatives, or etherial, alcoholic or ammonical stimulants may be given. Pilocarpin in $1\frac{1}{2}$ grain dose hypodermically may promptly secure a revulsion of blood to the skin, and at once overcome the chill and prove a most effective derivative from the pleura. Placing the legs in buckets of hot water, or the whole animal in a hot air bath will often act equally well. Packing the chest and even the abdomen in a blanket wrung out of very hot water and covering it closely by one or two dry ones, or, better still, by a rubber or other impermeable covering, will long retain both heat and moisture, securing free cutaneous circulation, and soothing in a most effective way the irritation in the chest. This may be maintained as long as requisite to relieve the patient, and then the body may be uncovered, a part at a time, rubbed dry and covered with a dry woolen blanket. Elastic circingles applied over the compress adapt it to the respiratory movements and any restriction in the movement of the ribs is beneficial by limiting the friction, pain and irritation in the pleura.

In the second stage, when inflammation has already set in, the same general measures of derivation toward the skin, and hot bath or soothing derivative compress are still demanded though they may be substituted by more stimulating derivatives. The bleeding of the patient into his own vessels is sought in various ways. On the continent of Europe stimulating embrocations (essential oils, ammonia and oil, mustard, etc.) are applied to the limbs.

In America and England similar agents are more commonly applied to the walls of the chest and dry cupping in the same region has been resorted to. Metallic cups with small mouths and having a capacity of about a pint each, have the air rarefied by plunging into each a spirit lamp or spill of burning wood, and, on its withdrawal, suddenly applying the mouth of the cup on the skin of the costal region, previously well coated with lard. Another form of cup is made with a tube and stop-cock in its otherwise blind end by means of which it is exhausted with a syringe after its mouth has been applied to the skin. In the absence of both, a narrow-mouthed glass tumbler may be employed, the air is rarified by inserting into it a burning spill of paper or wood for a few seconds and on its withdrawal the cup is instantly inverted on the skin. If the animal is very hairy or very thin it may be necessary to shave the part, and smear with oil and even to select a very narrow-mouthed cup. When applied, the cup is cooled with water or otherwise and owing to the partial vacuum the skin is strongly drawn up into it and the blood accumulates in and under the skin. It may be kept on for half an hour at a time and, with ten or twelve cups on one side, the patient tends to profuse perspiration, establishing a strong revulsion toward the skin, and great relief. In dangerous cases, three or four applications may be required in twenty-four hours.

Next to this the mustard application is perhaps the safest and most valuable. The best ground mustard (black by preference) is made into a very thin pulp with tepid or cold (never hot nor boiling) water and rubbed in against the hair so as to soak the surface of the skin; it is then closely covered with paper and with a rubber or other impervious covering or, in default of better, with a close blanket, and left on for two hours. By this time the skin should be thickened to the extent of at least a quarter of an inch and the derivation and relief will be very manifest.

Cantharides is sometimes used but, like most other severe irritants, is liable to induce sympathetic irritation in an already severely inflamed pleura, and thus to obviate all benefit. Cantharides is also liable, through extensive absorption, to irritate the kidneys. To counteract this, Bouley gave $\frac{1}{2}$ drachm doses of camphor with alleged good effect.

Some practitioners make local applications of hot water and of

aqua ammonia (confined) but unless very closely watched these are liable to destroy the hair follicles and produce permanent blemish. Vesicants should be used if possible before exudation. Laborde claims that, later, they increase the effusion.

The hot air, steam bath, and hot compress have the advantage over the more irritant derivatives that their action is from first to last soothing and free from all risk of inducing sympathetic irritation and yet as derivatives they are eminently efficient. A wet cloth or ice bag should be kept on the poll while in the hot air bath. Next to them in safety and efficacy comes dry cupping.

The irritant derivatives are often the most valuable, but must be used with great judgment. They are always dangerous when the pleural inflammation runs very high and when the local irritation and suffering are specially acute. Under such circumstances it is usually desirable to adopt other measures to moderate the severity of the inflammation, and to fall back on baths, compresses and cups until the irritation is alleviated before vegetable or animal vesicants are resorted to. In acute and severe attacks, these latter are especially applicable to the early stages before the inflammation has been fully formed, or after the stage of free effusion has set in.

With high fever and no benefit from hot local applications, cold irrigation or refrigerant compresses, to the walls of the chest, have proved useful, but considering the rôle filled by cold in causation and the suggested relation between pleurisy and rheumatism this is not to be followed as a general practice.

If the patient has been a hearty feeder and if there is evident costiveness, a purgative (aloës or sulphate of soda) is often desirable at the outset, but if the disease is of a low type this is always dangerous owing to susceptibility of the intestinal mucosa and it is safer to correct constipation by injections or at most by a pint of olive oil.

When the suffering is very acute and is aggravating the fever, a hypodermic injection of morphine will often greatly relieve, and even favor a revulsion of blood toward the skin, but as it tends to suppress the action of both bowels and kidneys it should be avoided unless it seems absolutely necessary, and above all should not be given by the stomach. Cocaine hypodermically may be used to relieve pain.

Both fever and suffering can sometimes be greatly relieved by large doses (2 drachms, 3 or 4 times daily) of salicylate of soda, which again suggests a close relation of the disease to rheumatism. Acetanilid or phenacetin may be used to fill the same indication. Tallianine may be used as in bronchitis.

Next come the questions of alkaline and diuretic treatment. Some cases do well if given nitrate of potash freely in the drinking water. Some prefer the alkaline diuretics, such as acetate of potash of ammonia, bicarbonate of potash or soda, biborate of soda, or the vegetable diuretics such as colchicum, squills, etc. Fraënkel found that, while comparatively ineffective alone, these proved most efficient (in man) when combined with cinchona or other bitter. The hint should be useful to the veterinarian. Diuretics in the stage of effusion should be pushed as far as the strength of the patient will warrant.

Friedberger and Fröhner recommend pilocarpin, and no agent produces an equal secretion from the natural emunctories and an equal tendency to reabsorption. It is, however, so profoundly exhausting that it must be used with the greatest judgment and caution.

Digitalis has often an excellent effect. Though not primarily a diuretic, it is a powerful tonic of the heart and circulation, and by increasing the blood tension it usually produces a free flow of urine. In combination with the diuretic salts it may be used from the first, but it is especially valuable after effusion and when attention must be given mainly to securing reabsorption. Care is demanded that we avoid its cumulative action, and in place of continuous large doses, a strong infusion applied over the loins will sometimes have a good effect. It may also be combined with bitters and even with ferruginous tonics in the advanced stages.

In combination with neutral salts and digitalis, iodide of potassium would seem to be indicated. Results, however, do not show a great superiority to other diuretics in favoring absorption.

Tincture of iodine, painted upon the chest over the affected parts, and repeated until tender, acts more or less as both a derivative and deobstruent. A liniment of iodide of potassium and soap is a convenient form of application.

The inunction of the chest walls with mercurial ointment has strong advocates both among physicians and veterinarians, and is combined in such cases with the exhibition of calomel internally. Unless the good effects are shown in a day or two it may well be abandoned.

When effusion becomes dangerous through excess, and in advanced cases when it fails to yield to medicinal measures thoracentesis is called for. (See under *hydrothorax*.)

ACUTE SERO-FIBRINOUS PLEURISY IN CATTLE.

Milch cows and work oxen most liable. Causes: damp buildings and locations, sudden transitions from heat to cold, exposure when fatigued, etc. Symptoms: rigor, reaction, cold horns and limbs, later hot, excited pulse, catching breathing, hyperthermia, 104° to 105° , tender chine and intercostals, friction sound, later dullness, creaking, weaker murmur. Subacute cases often tuberculosis, effusion unilateral, chronic cases. Lesions, as in horse with superficial marbling of lung. Treatment: laxative, warm drink, compresses, derivatives, sedatives, diuretics, heart tonics, diaphoretics, thoracentesis.

This is not common in young growing cattle, but is more frequent in milch cows and work oxen. It is due to the same causes as in the horse, and especially to chills when heated, damp buildings and locations, cold draughts between open windows or doors, and cold storms. The greatest danger comes from hot close stables, like many distillery stables, approximating to the temperature of the animal body and from which the stock are suddenly turned out of doors, or shipped by car or boat with a temperature near zero, and, above all, if furnished ice water to drink. Such animals, taking no exercise to increase the circulation and heat, are especially liable to shiver and contract illness. Rigors, too, are easily induced in animals standing in hot buildings, when, in connection with the cleaning, an adjacent door is thrown wide open, or two on opposite sides of the house. Working oxen heated with exercise and then exposed to extreme cold and compulsory inaction are endangered.

Symptoms. The attack is manifested by the same general symptoms as in the horse. The rigors are often very well marked, especially over the shoulder; the tenderness of the chine and

intercostal spaces is striking ; the breathing is catching but there is rarely the same restlessness as in the horse ; the bowels are costive, appetite and rumination impaired or suspended and the paunch is often distended with gas. The tenderness of the spine and intercostal spaces, the friction sound of the pleura, and the maintenance of the respiratory murmur and the normal resonance of the lung, become the ultimate diagnostic symptoms. The pulse may be 70 and upward, the temperature about 104° to 105°. In some insidious cases, indeed, the fever is very slight and besides the general wasting of the animal, the indications obtained by physical examination alone enable us to recognize the malady. Tuberculous pleurisy which is very common in cattle is to be suspected in such cases, and the tuberculin test should be applied.

Effusion is recognised by the dullness of the lower part of the chest up to a certain line, and often unilateral, by the softer pulse, by the dilated nostrils, or open mouth, the contracted facial muscles, the glazed eye, and anxious expression, by oppressed breathing and often by engorgement under the chest and in the limbs.

When the disease lasts over ten or twelve days it tends to pass into the chronic form. Or a chronic pleurisy of a subacute type may begin *de novo* and pursue an insidious and latent course.

If the disease commences as a subacute affection there may have been, for a month, capricious appetite, general ill-health and falling away before any other symptom is noticed. Now the breathing is manifestly excited, a small, short cough is heard at intervals, the pulse is accelerated but weak, and pinching auscultation and percussion detect unequivocal signs of pleurisy. From this the symptoms become more decided though for a length of time they are very slight, the animal meanwhile becomes increasingly emaciated, and perishes ultimately in a state of great weakness. Such insidious cases are always to be suspected of tuberculosis, and should be tested with tuberculin.

The *post mortem* appearances resemble those of the horse. The surface of the lung beneath the diseased portions of pleura, however, often presents a marbled appearance from the infiltration of the areolar tissue between the adjacent pulmonary lobules. The organization of the false membranes begins on an average about the tenth day. The effusion, reddish at first, becomes

clear after 10 to 20 days. Owing to the thicker mediastinum it is more likely to be unilateral than in the horse.

Treatment. The same general principles must be followed as in the horse. Bleeding can rarely be employed, partly because the disease so often assumes a subacute form, and partly because when first seen, considerable effusion has often already taken place and severe depletive measures are thereby contra-indicated.

A laxative dose (1 lb.) of magnesia sulphate, may be given in warm gruel, and the same means, by compresses, hot fomentations and counter-irritation, adopted, and the same sedative and diuretic medicines given, as in the horse. In the advanced stages and in the low types of the disease, the stimulating diuretics (sweet spirits of nitre, and liquor of the acetate of ammonia) and vegetable and mineral tonics are especially indicated. The diet in these last types must be nutritive, laxative and easily digested.

Tapping of the chest is equally applicable as in the horse, (*see Hydrothorax.*)

In the chronic forms, everything is to be done to support the general health whether by food, stimulants or tonics, and counter-irritants may be applied several times.

With animals in good condition it is often the most economical course to slaughter for beef at the onset.

SERO-FIBRINOUS PLEURISY IN SHEEP.

Causes : exposure, after clipping, washing in cold weather, alternations from hot buildings to cold fields, shedding of the wool. Symptoms, hyperthermia, troubled breathing and pulse with catching inspiration, tender intercostals, friction sound, and signs of effusion. Treatment, preventive, shelter, febrifuges in food or water, aqua ammonia to sides.

The causes of pleurisy in sheep may be largely included in the general statement—exposure. Cold washing, with exposure after clipping is especially injurious. Devieusart saw 300 cases of pleurisy and thirty deaths in a flock of sheep shorn in February. If kept secluded in warm buildings sheep may be shorn in mid-winter, but any reckless exposure, and any sudden reduction of the temperature of the building, is liable to be disastrous. *Scab* and other skin affections which lead to a shedding of the wool in inclement weather may also be the occasion of widespread attacks. Otherwise the causes are essentially those of the same disease in the larger animals.

The *symptoms* resemble those of pneumonia, but with the peculiar sharp, short arrest of the inspiration, and the marked tenderness of the intercostal spaces as above described. The cough is short, dry, hacking and frequent or suppressed as much as possible. Auscultation and percussion signs, corresponding to those found in other animals, are easily got in the newly shorn sheep. In the unshorn, the wool must be parted and a stethoscope employed. In thoracic effusion place the sheep in different positions and the flatness on percussion will always show at the lowest point.

The *treatment* is mainly *preventive* or when the disease is present, of a general nature applicable to flocks. A warm barn, with pure air, blanketing, wet compresses, to which may be added extract of henbane, and nitrate of potash in the drinking water, give examples of general medication. As a derivative, aqua ammonia and oil may be applied in lines on the chest exposed by parting the wool or generally on the shorn. Where the patient can receive the requisite attention, further treatment should be on lines laid down for cattle.

As in cattle it is often profitable to kill for consumption when first taken.

SERO-FIBRINOUS PLEURISY IN THE DOG.

Causes: exposure to cold, etc. Chill, reaction, disturbed breathing, catching inspiration, rapid, hard pulse, hyperthermia, tender chest, friction sound, later dullness at lower part of the chest in any position. Treatment: as in pneumonia, with antirheumatics and diuretics freely. Thoracentesis.

This is occasionally seen in the dog as the result of exposure, and like other diseases of the chest is easily recognized. It owns the same causes with pneumonia. Subacute and chronic cases are probably tuberculous or cancerous. It may occur by extension from adjacent organs, or from traumatism.

Lesions.—The pleura is congested, with arborescent redness, points of stasis or extravasation, cloudiness and opacity of epithelium, with swelling and active proliferation of the cells. Somewhat later, exudation takes place into and beneath the serosa and a reddish effusion from its surface collects in the pleural cavity, containing many red globules and granules. False membranes appear on the serosa, first as mere granular elevations, then as fine filaments, and as in other animals, forming first on the inflamed surface. False membranes are usually scanty as compared with horse or ox, arguing a lack of fibrine-forming elements in the effusion. The effusion may remain confined to one pleural sac, or invade both. When nodules form on the pleura the case may be adjudged tuberculous or cancerous.

Symptoms. There is first dullness, shivering and some excitement of respiration. To this follow the more acute symptoms, the hard pulse, the rapid, catching breathing, the animal standing, or sitting on his haunches, the open mouth, pendent tongue, the injected mucous membrane, the costiveness, but above all the tenderness of the intercostal spaces, the early friction sound on auscultation, the pain and normal resonance on percussion, the muscular twitching and the short, suppressed, painful cough. When effusion has occurred its amount may easily be estimated by turning the animal alternately on its feet, back and haunches, and observing how high the percussion dullness extends in these various positions.

The same *treatment* may be adopted as in *pneumonia*, with this difference that salicin may be given freely, derivatives pushed, and when effusion has taken place, active diuretics are especially indicated, and hence tincture of squills (a teaspoonful) may be made to replace the nitre. Digitalis, and potassium iodide may be usefully employed, and quinine, gentian or strychnia may be added. In advanced and obstinate cases, or where danger exists from rapid effusion, the liquid may be drawn off with a cannula and trochar as in other animals.

PURULENT PLEURISY (EMPHYEMA) IN SOLIPEDS.

Definition. Inflammation of the pleura, caused or complicated by the pus-forming microbes.

Causes. This may occur as a primary affection, the introduction of the pyogenic microorganisms being the essential microbial cause of the disease, or it may supervene on sero-fibrinous pleurisy, the pus germs finding their way later into the inflamed serosa. The possible channels of entry for these microbes are :

1st. *By the blood* as in strangles, glanders, contagious pneumonia, and influenza.

2d. *By the lymph channels*, as in fistulous withers, abscess of the intercostal or phrenic region, abscess in the abdomen, and even in strangles, glanders, contagious pneumonia, or influenza.

3d. *By rupture into the pleural sac* of abscesses in the lymph glands, bronchial, œsophagean, mediastinal, subdorsal or pre-pectoral.

4th. *Extension from pulmonary abscess.* Abscess in the lung in cases of pneumonia is quite liable to burst into the pleura, and the suppurative process in inhalation pneumonia will often extend to the serosa. Foreign bodies in the lung from whatever source may lead to purulent extension to the pleura.

5th. The penetration of a *sharp pointed body from the œsophagus*, or *rupture of the œsophagus*, in ill-directed efforts for the relief of choking will almost inevitably induce suppuration and empyema.

6th. *External wounds that penetrate the chest*, by shot, fork, nail, knife, spur, horn, tusk, pole, shaft, broken spike of wood or metal, or picket, railing or other object, open direct channels for the entrance of pus microbes, and empyema follows.

Bruises of the chest walls determining abscess may lead to escape of pus both externally and internally, and will not only infect but keep a channel open for further entrance of microbes. Similarly, injuries and necrosis of bones, (ribs, sternum,) may become occasions of empyema.

Lesions. The essential lesion is the presence of pus but as an accompaniment may be any one of the morbid conditions just enumerated. The purulent contents of the pleural sac may have one of various colors, dirty white, brownish, reddish brown, chocolate or greenish. When foetid there is usually gas (pyo-pneumothorax). In other cases it may have only a mawkish, heavy odor. The liquid is opaque and grumous, containing a profusion of red blood globules, pus globules and granules. It has largely parted with its power of coagulation so that the false membranes are few and spare. A yellowish pultaceous film covers the membranes and serosa generally. The surface of the pleura generally has a greenish hue and may be strewn with hard miliary nodules. The microbes of the liquid are not simply those of pus, but usually septic saprophytes as well, particularly when the foetor is strongly marked.

Symptoms. These are primarily those of the affection on which the empyema has supervened. Evidence of inhalation bronchopneumonia, abscess of the lung or chest wall, necrotic rib from fracture or otherwise, strangles with thoracic complication, a recent operation of thoracentesis, rupture of the oesophagus, or pneumonia or influenza with suppuration or gangrene, and developing specially violent and prostrating phenomena would assist in diagnosis. Temperature runs high, 104° to 106° F., but showing marked oscillations, the pituita is of a dark purple, the appetite lost, the intercostal spaces extremely tender, the discharge from the nose of a heavy odor and it may be foetid. There is marked implication of the nervous system, shown it may be in extreme dullness, prostration and muscular debility, or it may be in irritability, vertigo or even delirium. The animal loses flesh visibly from day to day. Percussion may show a lower area of flatness up to a given horizontal line and above this a tympanitic resonance.

Under such circumstances the puncture of the lower part of the chest with an exploratory needle will yield a purulent or even foul-smelling liquid which will confirm the diagnosis.

Course. The disease runs a rapid course usually terminating in death in three or four days.

Treatment. This is not at all promising with the disease fully developed. The main hope must rest on the disinfection of the

source from which the infection proceeded, and the evacuation and disinfection of the pleura invaded. Beside the treatment of the preëxistent and predisposing disease or injury, any injury to the walls of the thorax must be treated so as to evacuate all pus germs or other septic material at the earliest possible moment. Deep penetrating wounds must be probed, given free dependent drainage and packed with antiseptic gauze or subjected to antiseptic injections several times a day. Phlegmonous inflammations and forming abscesses are to be opened early and a free outlet allowed for the pus which might burrow onward toward the pleura. Disinfectants must be freely used: mercuric chloride (1:1000) when the drainage is free: in case of a very extensive cavity a 5 per cent. solution of creolin or lysol may be used. In all such cases it is to be considered that it is not the antiseptic alone that proves effective. The protective action of the tissue and blood leucocytes and of the embryonal cells multiplying in the affected tissues is of itself sufficient to deal successfully with a slight local invasion, and our assistance in the way of antiseptics is to limit and prevent the dangerous excess of the invading microbes. In caries of a rib, resection may be demanded conducted with similar antiseptic precautions.

When, on the other hand, empyema is already present, the evacuation of the liquid from the pleural sac is the first consideration. This may be accomplished by aspiration, by the cannula and trochar or by simple incision and drainage. In all alike the greatest care must be taken in the disinfection of the skin in the seat of incision or puncture, of the hands of the operator, and of the instruments. We are dealing with an already infected part but it is possible to add other and even more deadly infections to the already existing ones. Shave the surface, wash thoroughly with soap and boiled water, then douche with mercuric chloride solution (1:500). Deal with the hands in the same way and boil all instruments. The incision must be at the front of the ninth rib close to its union with its cartilage, and to avoid possible risk of striking the diaphragm, the point of the aspiration needle or trochar may be directed forward and upward after it has passed through the intercostal muscles. Having drawn off the septic or purulent fluid, the pleural cavity may be washed out with a saturated solution of salicylic acid or a 3 per cent. solu-

tion of boric acid, or pyoktanin 1:2000 solution may be used. In case of simple incision this is done at the same point, with corresponding precautions, and the cavity is to be disinfected in the same way. Then the opening may be dusted with iodoform or aristol, and covered with sublimate gauze or cotton sustained by a bandage. In case of necessity an aseptic drainage tube may be used.

The greatest attention must be given to secure generous nourishment, pure air and genial temperature. Internally quinia and calcium sulphide may be given.

PURULENT PLEURISY (EMPHYEMA) IN CATTLE.

This may occur in cattle from the same kinds of injuries as cause it in solipeds. Broken ribs followed by necrosis or caries, penetrating wounds of the thoracic walls from horns, tusks, etc., bruises and abscesses of the chest walls, sternal or costal, perforations of the gullet by sharp pointed bodies or by whips or other unsuitable bodies in ill-directed attempts to relieve choking, come under this head. A special cause is found in the migration of sharp-pointed bodies from the reticulum into the chest and usually toward the heart. It may be the result of rupture into the pleura of abscesses in the lung, bronchial glands or elsewhere in the chest, and still more commonly from softened and caseated tubercle, glandular, pulmonary or pleural. Advanced lung plague with suppurating sac enclosing sequestrum, and pyæmia from distant purulent or septic centers of infection may become causes. Septic metritis after calving has been specially incriminated.

Lesions. As in horses these are as varied as the primary cause of the trouble. The effused liquid is opaque, grumous, with many cell organisms and much granular matter, usually very foetid, and of a color varying from a dirty yellow or grayish white to a deep dark red or even black. The serous membrane is congested or it may be pale. In cases due to tuberculosis the tubercles may be present in all conditions from the recent firm red nodule, to the caseated mass and vomica. The liquid in this case contains the caseated débris and shreds of decomposing membrane, and, in common with the tissues, the bacillus tuberculosis.

Symptoms. There are the symptoms of pleurisy, complicated by those of the wound or injury; the indications of a foreign body advancing through the chest toward the heart, especially tympanies of the rumen, foetid regurgitations, a line of flatness on percussion from the reticulum forward with a mucous or bubbling râle, an irregular action of the heart, and pasty swelling around the sternum; and, finally, the crucial test of drawing off a little liquid with a hollow needle and subjecting it to examination. In cases due to uterine suppuration the vaginal discharge will guide; in pyæmia the discovery of multiple abscesses; in tuberculosis the disorder of the digestion, the presence of tubercles in the throat, the percussion and auscultation indications of tubercles in the lungs, the swelling of the lymph glands in the abdomen, or in the intermuscular groups,—præpectoral, prescapular, prefemoral, inguinal, etc., and, finally, the result of the tuberculin test.

Treatment. Beside the general treatment of the primary cause, the removal of the infected effusion with the same antiseptic precautions as recommended in the horse will be required. Also the same general dietetic and hygienic measures. The case is not hopeful in any instance, and is especially bad in tuberculosis. The most hopeful cases are such as supervene on simple penetrating wounds, where the infection has not yet seriously advanced, and the strength of the patient is well maintained. But these are just the cases in which a certain salvage can be secured by turning the animal over to the butcher.

PURULENT PLEURISY (EMPHYEMA) IN SHEEP.

In sheep the usual cause is inhalation broncho-pneumonia, hemorrhagic septicæmia affecting the chest, and traumas. The *symptoms* are the counterparts of those of the ox, and the *general treatment* the same.

PURULENT PLEURISY (EMPHYEMA) IN SWINE.

The strong indisposition of the pig to pus infection serves to protect it against empyema, and a further measure of protection is found in the usually thick panniculus adiposus. Still in exceptional cases purulent and septic infection in the lungs will in-

vade the pleura, and tubercles in pleura or lungs or bronchial glands will burst into the pleural cavity. Again sharp-pointed bodies swallowed will penetrate the gullet and start infection in the pleura, and solid bodies obstructing the œsophagus may be forced through its walls, to enter and infect the serosa. Such cases are not encouraging to treat, but simple penetrating wounds may be more hopefully dealt with than in ruminants and solipeds.

PURULENT PLEURISY (EMPHYEMA) IN DOGS.

Dogs are especially subject to inhalation pneumonia and septico-purulent extension to the pleura. Distemper too, when implicating the lungs, is very liable to extend into the pleura. Otherwise, purulent pleurisy in this animal is very liable to come from mechanical injuries. Kicks by horses, cattle or men, and blows with heavy clubs which fracture the ribs and lacerate the pleura and even the lung, producing at the same time a local inflammation which smooths the way for the entrance of pus germs, are common cases. Penetrating wounds by shot, stable forks, knives, horns of cattle or deer, and the tusks of the boar are further causes. Sharp-pointed bodies swallowed (pins, needles, nails, and above all sharp pieces of bone) are liable to be arrested in the gullet and to make their way through to the mediastinum and pleura. One case is recorded of even an ear of a graminea passing forward through the diaphragm with its base directed forward and its glumes feathered back like an arrow so that it could not recede (Siedamgrotzky), and another in which a spike of rye had perforated the bronchium and lodged in the pleural cavity with resulting septic pleurisy. I have known part of a sheep's vertebra to be lodged in a dog's œsophagus for two months, the animal being sustained meanwhile by the milk that passed it. Such obstructions cause inflammation and infection proportionate to the size, the sharp points, or roughness on the surface.

Other cases of purulent pleurisy are the result of ruptured tubercles, cancers and other morbid products in the chest.

Lesions. Pleurisies resulting from inhalation pneumonia have the general lesions of the bronchia and lungs that mark that complaint, together with an excessive, fœtid effusion of a dark brown, chocolate, or coffee-grounds color, and extensive, loose

coagula of the same shades. The liquid contains flocculi, granules, and debris which fall to the bottom when allowed to settle in a bottle. The affected portion of the lung is infiltrated, consolidated, grayish, greenish or brown, and easily broken down under the finger. In simple penetrating wounds the effusion is white or yellow and in small amount. In tubercle or cancer these morbid conditions are easily detected on the surface of the lung or elsewhere.

Symptoms. Fever runs high, the temperature rising to 105° F. but subject to marked fluctuations. Pulse is rapid, breathing difficult, animal dull, weak and prostrate and the objective signs indicate a lower collection of liquid effusion with a resonance showing a formation of gas above. The chest walls are dilated on one side or both, and extremely tender to touch or percussion, the back is arched and the abdomen often tympanitic. Retching or even vomiting is to be looked for. The primary trouble, wound, choking or inhalation, will often complete the diagnosis. If not, a fine exploring needle, passed into the chest, will show the nature of the contained fluid, creamy white, or dark brown, or green and foetid.

Prognosis. From inhalation-infection this is very fatal, but from a simple wound of the chest wall, recovery may be hoped for, the resistance to pus infection being greater in the dog than in the larger herbivora, though less than in the pig.

Treatment. In case of external wounds probe and if necessary open a free exit for any pus; use disinfectants as injections or on sterilized cotton, in case of abscess make an early free opening so that discharge will be unimpeded, and inject antiseptics. In case the pleural sac is filled septico-purulent fluid, and in the absence of irremediable and fatal lesions of other kinds in the chest, draw off the liquid by aspirator or trochar and cannula with all due antiseptic precautions, and irrigate the cavity with a saturated solution of salicylic acid or a three per cent. solution of boric acid, or again it might be completed with a weak (1:2000) solution of pyoktanin. Absolute cleanliness, pure air and generous feeding are important. Quinia or strychnia may be given in form of pill, and small doses ($\frac{1}{6}$ grain) of calcium sulphide may be given several times a day.

DRY PLEURISY IN THE HORSE AND OX.

Definition. This is a form of pleurisy in which the exudate coagulates at once on the inflamed surface, and no appreciable amount of liquid is thrown out. For this reason, probably, the infection does not extend widely and it is manifested by clear, glistening, white patches, varying in size from one to six inches in diameter, and usually firm and resistant. In its lactescent color it is in marked contrast with the rosy red of the adjacent serosa. Occurring mainly in old animals, it implicates by preference the outer side of the lung in the region of the heart or near its upper border, and often establishes an adhesion between this and the ribs, the diaphragm, or in cattle between lobe and lobe. In some cases the membranes are shreddy as if they had been torn from their adhesions. If recent these membranes are granular or fibrillated; if of older standing they have become firm, fibroid and tough. They may bind the lung closely and immovably to the ribs, or they may be loose, mobile, and allow the free movement of the one on the other.

Causes. Many cases appear to be the result of a constitutional condition, like rheumatism, occurring at points where external injuries could have little effect (under the muscular shoulder or back). Yet Cadeac draws attention to cases in which fistulous withers have been attended, as shown *post mortem*, by inflammation and the formation of false membranes on the pleura just beneath. Chronic pneumonia occurring in old animals seems to be especially subject to the complication of dry pleurisy. Again, tumors in the lung and comparatively inactive pulmonary tuberculosis, are subject to this pleuritic complication. Yet dry pleurisy appears at times to come from the same general causes as sero-fibrinous pleurisy, acting perhaps on a system with a special insusceptibility.

Symptoms. These are in the nature of things obscure. The disease is usually circumscribed in area, and limited to points where the movements of the lung are most restricted, and the surface most deeply covered by the bones and thick muscular masses of the shoulder or back, so that in some cases, in the absence of exertion, there is no marked disturbance of the breathing. In other cases, however, there is the short, abruptly cut

off inspiration of pleurisy, and even a double lifting of the flank in the expiration and, finally, a distinct friction sound coincident with the completion of the inspiratory act. The friction sound is the one pathognomonic symptom, hence it should be carefully sought for. Later when the false membranes and adhesions have become partially organized, their stretching may give rise to creaking sounds in the movements of the chest. There is also the usual tenderness of the intercostal spaces in the region of the exudate, demonstrable when that point can be reached. There is no appreciable dullness on percussion, and no indication of pulmonary crepitation in uncomplicated cases. Therefore, most examples of dry pleurisy and false membranes have been found *post mortem*, and without any previous recognition during life.

Prognosis. Dry pleurisy is in its nature circumscribed and without immediate danger to life. The active inflammation and fever, tend to recovery, but there may remain more or less compression of a portion of the lung with imperfect filling of the air cells, and in addition adhesions between the lung and the walls of the chest, which together impair respiration so that both wind and pace are interfered with.

Treatment. Opportunity for treatment is usually denied for lack of a correct diagnosis. When diagnosed, the affection may be met by damp compresses, hot fomentations, dry cupping, and even stimulating embrocations and mild blisters. Internally, soda sulphate may be used daily to keep the bowels open, and small doses of sodium or potassium bicarbonate may be used to lessen the formation of false membranes, but the diuretics so much in demand in sero-fibrinous pleurisy are here quite unnecessary. As the inflammation subsides, bitters and iron may be given to build up the health, and a generous diet and open air exercise may be gradually employed.

PULMONARY EMPHYSEMA IN CATTLE.

Causes. This may occur in the lobular or interlobular form from hard work in the yoke or from violent exertion of any kind, such as straining against hobbles when *cast* (Anacker), a stampede, the continuous bellowing of an animal shut up alone, the persistence of a cough from disease of the throat or lungs (pharyngeal or pulmonary tuberculosis, tumors in the throat, lungs or mediastinum, lung worms, dusty food, inhalation broncho-pneumonia, pleurisy from foreign body or other cause, heart disease, lung plague and its sequelæ, Rinderpest, etc.), swill-stable feeding, tympany of the rumen, traumas of the trachea or bronchia.

Lesions. These resemble those of the horse, the affected lobules standing out like small comparatively bloodless bladders on the surface of the lung, more particularly near its lower border and in the forward lobes. There is this marked addition in interlobular emphysema that the great profusion of connective tissue between the islets of lung tissue is replete with air and stands out clearly, marbling the lung as in lung plague, and even leading to mistaken diagnosis. This has led to the confounding of lung plague and rinderpest in former times. The lines of blown up interlobular connective tissue differ from the liquid-infiltrated specimen of lung plague, in that it is silvery or dark instead of white or yellow, it crackles when manipulated and it collapses promptly when pricked.

Following the abundant connective tissue in cattle the emphysema is liable to extend to the roots of the lungs, the mediastinum, the connective tissue at the entrance of the thorax, the lower part of the neck, shoulders, sternal region, walls of the chest and loins. It may even penetrate the diaphragm by the side of the œsophagus and reach the peritoneum.

Symptoms. These are less marked than in the horse. The breathing is rapid, it may be panting after active exertion and in some instances there is a double rise of the flank with a motion of the entire trunk in expiration. The most conclusive evidence is obtained in the drum-like resonance on percussion over the affected part of the lung, mostly below and in front. The diagnosis is often assisted by the presence earlier of some inflammatory affection of the lungs: bronchitis, pneumonia, broncho-

pneumonia, pulmonary tuberculosis, etc., which has determined the alveolar distension and rupture. In inhalation-bronchitis above all, there is liable to be the subcutaneous tympany in the breast, or on the sternum or shoulder. Auscultation may detect modified sounds, subdued or exaggerated respiratory murmur, wheezing, sibilant sounds, or even crepitation.

In very extensive cases there may be extreme dyspnoea, hurried breathing, violent lifting of the flanks, open mouth, pendent bluish tongue, and fits of coughing, with tympany (Sarradet). This form, which occurred in paroxysms, several times a day and continued to appear for fifteen days, when it entirely subsided, was manifestly something more than simple emphysema though that was an accompaniment of the affection.

Treatment. Prevention must be sought by obviating inflammations of the air passages and all causes of violent and persistent cough. In the same way extreme heat, close buildings, exposure in the hot sun, and violent exertions should be avoided. To give better tone to the vagus nerve a long course of arsenic may be given with or without nux vomica and digitalis. Indigestible food, overloaded stomach and tympanies should be corrected. Laxatives, and moderate, easily digested, laxative diet may be given, and free access to abundance of pure water.

PLEURO-PNEUMONIA. BRONCHO-PNEUMONIA.
BRONCHO-PLEURO-PNEUMONIA

Though we often meet with typical forms of *bronchitis*, *pneumonia* and *pleurisy*, it is much more common to find them combined more or less with each other. Thus, combined inflammation of the bronchial tubes and pulmonary substance is frequent ; inflammation affecting both the lung and its investing pleural membrane is no less common ; and cases are seen in which all three structures are involved. These conditions are to be recognized by the presence of the symptoms of both the coexisting maladies, but particularly by the indications furnished by touch, auscultation and percussion. The predominance of one disease over another will decide the nature of the treatment which will be adapted to the peculiar character of each case whether *mainly bronchitic*, *pneumonic* or *pleuritic*. It is these mixed cases that test the ability and judgment of the practitioner, as he must carefully individualize each case, ascertain the different parts affected, the grade of the inflammatory action, the nature of the attendant fever, the presence or absence of epizootic influence, infection, etc., and having all these conditions in view, must apply remedial measures accordingly.

It must be evident that particular directions cannot be supplied for all of these cases. General principles only can be inculcated and their adaptation to the varied phases of different cases left to the judgment of the student.

HYDROTHORAX.

All animals liable. Causes : pleurisy, obstruction to pulmonary or intercostal veins, heart disease, Bright's disease, anæmia, parasitic or otherwise. Effusion reddish gray or clear straw color. Inflammatory and dropsical. Symptoms : troubles of respiration, as a secondary disease complicated by dropsies elsewhere, signs of hydrothorax without fever, shedding of hair. Treatment : diuretic, tonic, derivative, thoracentesis, trochar and cannula or aspiration, point of election for puncture, method, asepsis, drainage by aspirator, or into an antiseptic solution, eligible cases, dangers attending thoracentesis, shock, rupture of false membranes and lung, infection of pleura, injection of antiseptics.

Hydrothorax or *water in the chest* is common to all domestic animals. It is, as we have seen, one of the most ordinary results of *pleurisy*, and may persist long after that disease has disappeared. It likewise occurs independently of inflammation as a dropsical effusion. Thus, when the return of blood by the bronchial, pulmonary or intercostal veins, is hindered by any cause such as tumors in the bronchial glands or subvertebral region, a passive effusion may take place through the coats of the vessels. In imperfection of the mitral valves the regurgitation of blood in the pulmonary veins during each cardiac systole equally causes such transudation. Chronic disease of the kidneys (Bright's disease), with the retention of effete and injurious materials in the blood, leads to dropsy of the chest, as in other parts of the body. Again, in many debilitated conditions parasitic and otherwise, with a tendency to general dropsy, the chest participates and a collection of fluid takes place in the pleura.

The nature of the contained fluid will vary according to the conditions in which it has been effused. If the result of inflammation, there are the different stages already indicated : *first*, of a yellow citrine color, or red from contained blood ; *second*, grayish and muddy, either from contained pus or other changes taking place in the fluid ; and *third*, clear, limped and translucent as seen in the later stages. If merely a dropsical effusion, the fluid is watery, clear and translucent or with a slight straw color. The inflammatory effusion contains fibrine or fibrinogenous elements, is associated with the formation of false membranes, and though it may remain fluid so long as it is retained

in the chest, it coagulates rapidly when withdrawn. The dropsical effusion rarely contains fibrine, and then only in very small amount, and it does not coagulate when drawn off from the chest. The inflammatory effusion usually contains a greater proportion of common salt, phosphates or albumen than exist in the blood, and floating granules, particles of cell forms, none of which conditions characterize the dropsical effusions. The most prominent feature of the inflammatory infusions is thus seen to be their power of coagulation, by virtue of the contained fibrine, when exposed to the air.

Symptoms. When a sequel of pleurisy, it is manifested by the symptoms already mentioned under that head as indicating the occurrence of effusion.

The dropsical cases may come on rapidly and present all the signs of troubled respiration together with the results of auscultation and percussion that characterize rapid inflammatory effusion but without the fever and acute symptoms of pleurisy. More usually it comes on insidiously, the lung accommodates itself to the gradual increase of the fluid, and it is only when the accumulation has become excessive that the symptoms become prominent. In heart or kidney disease the filling of the legs and infiltration of the eyelids and of the skin beneath the chest and abdomen are precursors or early concomitants of the disease, but in all cases the accumulation in the chest is to be measured by the height of the line of dullness on percussion and the extent of chest surface giving forth no respiratory murmur on auscultation. As the liquid rises on both sides of the chest, as it always does in such cases in the horse, the breathing becomes short and labored, being chiefly effected by the action of the diaphragm and the flanks—the ribs moving only slightly. The nostrils are widely dilated with each breath. The previously existing want of vigor and energy, the weak pulse, the poor appetite and the pallor of the mucous membranes become aggravated; the animal becomes very weak and prostrate, the loins insensible, the permanently tucked up flanks labor tumultuously, the loins rise in inspiration, the face is pinched and haggard, the eyeballs glazed and protruding, and death is preceded by the same general symptoms as in rapid effusion after pleurisy. A prominent feature of this, as of all dropsical affections, and one usually seen in the hydrothorax of

inflammation as well, is the ease with which, even at an early stage of the disease, the long hairs of the mane and tail may be pulled out. In many cases they come out in handfuls when the comb or the fingers are passed through them.

Treatment. The treatment must be of the actively diuretic kind recommended for the effusion of pleurisy. It is modified, however, in one respect. The inflammatory action having subsided or nearly so and the condition being now essentially one of weakness a free use of tonics is demanded. Many a patient dies in such circumstances from the actively depletive treatment to which it has been subjected and the want of attention to its need of generous diet and other support. The agents prescribed for the advanced stage of pleurisy may be given, or the digitalis or other diuretics and bitters may be combined with iodide of potassium in one or two drachm doses, the amount being apportioned to the strength of the animal. Iron in the form of sulphate, perchloride or iodide may be freely given combined with gentian, quassia, or other vegetable tonic, and, above all, a liberal and easily digested diet must be allowed. Good will sometimes result from repeated applications of strong iodine ointment to the sides with active friction.

When the condition is dependent on diseases of the heart, kidney or other organ, these must be attended to according to their special requirements.

Disconnected from such complications, hydrothorax will often give way to an active treatment similar to that indicated above. In some cases, however, our only hope of even temporarily prolonging life lies in the operation for drawing off the fluid.

Tapping the chest or as it is technically called, **thoracentesis** or **paracentesis thoracis**, has proved sufficiently successful in the lower animals to warrant its continuance in cases that resist other modes of treatment. It is highly probable that the larger proportion of unsuccessful cases is due in great part to the hopelessly advanced stage at which the operation was resorted to, to the insufficient precautions adopted in its performance, and to the want of appropriate dietetic and medicinal treatment. Dr. Bowditch's treatment by *paracentesis* saved, in the human subject, at the rate of about two patients in five and we ought by availing of similar precautions to reach the same standard.

The cannula employed in veterinary practice is a silver tube two inches in length, a quarter of an inch in diameter and furnished with a shield of the same metal at one end. The trochar by which it is introduced is of steel or brass. To carry out Dr. Bowditch's system, the operator must supply himself with a syringe of a somewhat smaller bore and an intermediate brass piece of a size adapted to fit accurately into the cannula and supplied with a stopcock. By an instrument of this kind, the fluid can be drawn off by means of the syringe, without any risk of the introduction of aerial germs which always tend to induce suppuration and even a putrefactive decomposition in the contained fluid.

The point selected to operate on is, in the horse, ox or dog, in front of the anterior border of the ninth rib, at its lower end or close to its union with the cartilage. The point of the trochar should be directed slightly upward and forward to avoid the possibility of injuring the diaphragm. The skin is first rendered aseptic by shaving, followed by a thorough soapy wash and a free use of mercuric chloride solution (1 : 500). It is then incised with a lancet, then drawn aside that the wounds in the skin and muscles may not correspond after the cannula has been withdrawn. The trochar is then pushed steadily through the intercostal space till all obstruction has been overcome, when it may be concluded that the pleural sac has been reached. The trochar is now withdrawn and the fluid allowed to flow from the cannula until there is presumably some risk of the introduction of air, when the brass piece is to be applied and the remainder drawn off with the syringe or aspirator. As a substitute for the aspirator, a caoutchouc tube, eighteen inches long, put on a cannula or needle and having its lower end plunged in a solution of boric acid, will prevent the entrance of germs. A probe has often to be introduced to prevent plugging of the cannula by floating false membranes, and a new puncture in a different place may be necessary. In the case of excessive accumulation it is often advisable to draw it off at two operations, as recommended in large abscess of the pleura and for the same reasons. The need for such a precaution will be understood when it is stated that in bad cases the chest contains as much as six or seven ordinary stable bucketfuls of the liquid. If, however, it is limited in amount it may be all withdrawn at once.

The most successful cases in the horse have been upon young, vigorous animals, from four to eight years old, during the first month of illness, and where the pleurisy has been confined to one side.

Dr. Bowditch lays down the following rules for the adoption of paracentesis in man (*Clinical Medicine*, by Prof. W. T. Gairdner) :—

“ I now never operate unless I find some distention or rounding out of the chest, and filling up of some of the intercostal spaces, so that the chest presents a uniform curve, and not alternate depression and elevation as in the healthy chest. I operate under the following circumstances when I feel certain there is fluid :

“ 1. When there is *severe permanent dyspnœa*—orthopnœa—however acute the disease if I find fluid filling the pleural cavity, or nearly filling it.

“ 2. When there are occasional attacks of orthopnœa threatening death even if there be not sufficient to fill more than half of the cavity. If the fluid seems to be the cause of the dyspnœa I operate, because occasionally I have lost a patient while waiting for more extensive physical signs. This rule I apply to acute and chronic cases.

“ 3. I use the trochar after three or four weeks of ineffectual treatment without any absorption being produced.

“ 4. In chronic idiopathic hydrothorax, a latent pleurisy with simply physical signs to indicate *extensive* effusion, but when the rational signs are either very slight or none at all save a general malaise and weakness.”

The use of iodide of potassium and vegetable and mineral tonics must be perseveringly kept up and the strength further supported by a generous diet, to secure the animal against the dangers of extreme prostration, of suppuration, or other undesirable conditions of the exuded product.

Among the dangers attending thoracentesis, are fainting as a result of shock on the sudden withdrawal of so much liquid, rupture of the false membranes, and even of the enclosed lung tissue or of blood vessels, under the sudden expansion of the partially collapsed lung confined by the investing false membrane, and the introduction of pus or septic germs into the pleural cavity. To obviate the first named dangers tight bands (circingles) around

the chest will give support and limit sudden expansion. In case of excess of liquid the withdrawal of one-half or two-thirds at a time will allow opportunity for accommodation. Hæmorrhage may be met by the internal use of chloride, sulphate or nitrate of iron, matico, hamamelis or tannic acid, and a weak solution of boric acid or other antiseptic agent may even be injected in small amount into the pleural cavity.

In obstinate and chronic cases the injection of a weak solution of iodine and iodide of potassium is often of service. In other cases a normal chloride of sodium solution (previously sterilized) may be introduced as soon as a partial evacuation causes uneasiness, and by a succession of such evacuations and injections the residuum liquid may be rendered clear and largely aseptic on a single occasion.

In the smaller animals the selection of the most dependent part for insertion of the trochar is not so essential, as the body may be turned to facilitate the drainage.

On completion of the operation the wound may be again treated antiseptically and coated with aristol or collodion.

PNEUMOTHORAX. AIR OR GAS IN THE PLEURA.

Causes: decomposition of liquid effusion, perforation from a bronchium, the stomach, a thoracic wound. Symptoms: metallic tinkling, splashing, succussion, drum like resonance, suppressed respiratory murmur, distance of cough sound, distress, anxiety, dyspnoea, bulging, intercostal spaces, sometimes a wound. Treatment: closure of wound, calmatives, aspiration of gas. Treatment for pleurisy.

The collection of air or gas in the cavity of the pleura has already been noticed as co-existing with liquid effusion in some cases of advanced pleurisy. It may arise from other causes, among which may be noted: 1. When a mass of pulmonary tubercle connected with a bronchial tube has opened into the pleural sac. 2. When a communication has been established between the pleural cavity and the alimentary canal, as in combined rupture of the stomach and diaphragm, or of the double colon and diaphragm. 3. When a rib is fractured and the broken end penetrates the lung tissue and opens into one or more small bronchial tubes. 4. When a wound has been inflicted penetrating the walls of the chest and forming a valvular orifice through which air is drawn inward during each inspiratory act, but out of which it cannot pass when the thorax collapses.

The amount of gas present may be extremely slight, or in a case such as that from a valvular wound it may cause complete collapse of the lung, filling up the entire half of the thorax and bulging into the opposite half.

The *symptoms* are often very obscure. If with liquid, the metallic tinkling after rising, in small animals the splashing when shaken, and the other sounds of auscultation and percussion will point it out, as described under *pleurisy*. In the case of a broken rib, the distortion, swelling and tenderness, will lead to suspicion. A penetrating wound will be sufficiently evident, and in the case of tubercle, previous cough and ill-health will have been manifest.

The specific signs of uncomplicated pneumothorax are: 1st, a drum-like resonance on percussion over the seat of the gas, usually at the upper part of the chest; 2d, a partially suppressed or distant respiratory murmur over the same area; 3d, a muffled or suppressed sound of the cough; 4th, sometimes, especially if the gas is abundant, prominence of the chest on that side; 5th,

there are also more or less distress and anxiety, difficult breathing, quick, weak, rapid pulse, and other signs of illness.

Some cases of this kind recover spontaneously or with the withdrawal of the liquid effusion, with which they are associated ; in traumatic cases the wound is sometimes sealed up by a pleuritic exudation which here becomes a curative process ; while in some examples of valvular wound of the lung or walls of the chest, death may ensue in a period varying from a few minutes and upwards to weeks.

Treatment is limited to the prevention of the ingress of air through an external wound if that exists ; the employment of opiates and other agents to moderate attendant suffering ; to measures calculated to moderate the intensity of resulting pleurisy, and, in cases where there is imminent danger from accumulation of gas, to the puncture of the chest and the careful withdrawal of the gas by aspiration. If necessary, sterilized air may be made to replace in part the aspirated gas.

CHRONIC PLEURISY.

Animals liable. Causes: irritation through effusion and exudate acting on susceptible pleura, or by other disease products in lung or pleura. Unhygienic surroundings and management predisposes. Frequent chills in cold water. Symptoms: unthriftiness, easily blown, fatigued or sweated, cough paroxysmal under exertion, pallid mucous membranes becoming congested on slight exertion, difficult breathing when recumbent, percussion and auscultation signs of pleurisy and hydrothorax. Lesions: great liquid effusion, clotting on exposure, with much albumen and cell-forms. False membranes partially organized. Treatment: tonic, diuretics, derivative, diet nourishing, counterirritants, paracentesis.

In all domestic animals acute pleurisy may merge into the chronic form, the irritation being maintained by the presence of the residuum liquid and the false membranes and adhesions which interfere with the free dilatation of the chest. The pleura too, having been once inflamed, retains an increased susceptibility to such disturbing conditions. In other cases the affection is symptomatic of other chronic affections, as tuberculosis, glanders and neoplasms of various kinds. It has been seen especially in old, weak and debilitated subjects, kept in confined, impure stables or habitually exposed to undue cold and damp. Hence dairy cows in unhygienic conditions, and hunting dogs, which plunge in water when heated, are among the most frequent victims.

Symptoms are often obscure. The affected horse may be bright and lively, showing little respiratory disturbance unless under exertion. Yet there is a general appearance of unthriftiness, with erect, dry hair, hidebound, and a small, dry cough. Under work there is hurried breathing, early exhaustion, ready perspiration, and aggravation of the cough which then occurs in paroxysms. Auscultation and percussion give characteristic signs according as there may or may not be false membranes or effusion at particular points. It is usually bilateral in horses, unilateral in other animals.

In cows in addition to the corresponding symptoms, there is pallor of the mucous membranes when at rest, quickly transformed into congestion under exercise, suppression of the milk, and weak heart beats unless when excited. In the advanced condition the animal has difficulty of breathing when recumbent on the

sound side, and subcutaneous infiltration is felt or seen beneath the sternum or in the limbs. The affected side shows an increased dimension, vertical and longitudinal, of the chest, and the intercostal spaces in their lower part bulge out and fluctuate.

In cows and indolent animals there may be a quiescent condition or very slow progress, but any violent exertion is likely to give a sudden stimulus to the morbid process.

Lesions. The liquid effusion, usually unilateral, except in the horse, may amount to 40 quarts in the latter animal, 30 quarts in the ox (Rigot), and 5 to 6 quarts in the dog. Unless there has been a recent sudden accession of inflammation it is of a pale straw color, with, in the dog, a slight rosy tinge. It clots loosely on exposure to the air and contains a large amount of albumen and few cell-forms. The false membranes are thick and white at some points and red and vascular at others. In the main they are completely organized. The lung is more or less collapsed and the right heart dilated, with attenuated walls.

Treatment must be, in the main, tonic, diuretic and derivative. Food must be nourishing, digestible and in liberal amount; diuretics and bitter tonics with digitalis and, (if there is little fever) preparations of iron are to be pushed as far as the strength will allow; and the counterirritants applied to the sides of the chest a number of times in succession. Iodides may be used internally and externally, and *paracentesis* must be employed unless early improvement is manifested.

PLEURODYNIA.

Definition. Symptoms : stiffness, pointing of fore limb, catching inspiration, tender intercostals, less fever, cough, and hardness of pulse than in pleurisy, no friction sound nor sign of pleuritic effusion. Treatment : anti-rheumatic, derivatives, colchicum, alkalies, salicylate, salol, phenacetin, warm (steam) bath, warm building and clothing.

Definition. Rheumatism of the intercostal muscles. This has been occasionally observed in the horse, and is liable to be mistaken for pleurisy, which it closely resembles in its symptoms. There are the same stiffness of the fore limb on the affected side, the same short breathing, the same fixed and inactive appearance of the ribs, and the same extreme tenderness on pressure as in pleurisy ; but the high type of fever, the cough and the full hard and accelerated pulse are usually absent ; the tenderness tends to shift from one point to another, there is no shivering nor friction sound in the early stages, and no subsequent absence of sound and deadness on percussion over the lower part of the chest, as result from effusion. When associated with fever it is very difficult to distinguish from pleurisy, and its recognition can only be made by the physical signs just mentioned.

Treatment. This must be the same as in rheumatic attacks in general. Rub the chest actively and repeatedly with a mixture of equal parts of spirits of turpentine, laudanum and olive oil, give $\frac{1}{2}$ drachm doses of powdered colchicum daily and bicarbonate of potass freely in the drinking water. Or give four times a day 2 to 4 drachms of salicylate of soda, or 1 drachm of salol, or phenacetin. A warm building and warm clothing are essential elements in treatment.

BRONCHIAL ASTHMA IN THE DOG.

Definition. Pathology, neurotic origin, bronchial spasms, swelling of mucosa, fibrinous inflammation of bronchioles, Berkart's streptococcus, irritants formed in indigestion, overfeeding, inactivity, plethora, constipation. Symptoms: obesity, sluggishness, recurrent paroxysms of dyspnoea, hard cough, tense abdomen, constipation, piles, depilation of skin, tartar covered teeth, foetid breath. Retching, vomiting a glairy mucus, emaciation may follow. Lesions: emphysema, fatty deposits in mediastinum, old standing diseases of the heart, lungs and digestive organs. Treatment: antispasmodics by lungs or rectum, stramonium, nitre fumes, emetic, purgatives, vegetable diet, exercise, sedatives, blisters. Asthma in the horse.

Definition. A neurotic affection mainly affecting the pneumogastric nerve, and leading to paroxysms of stenosis or constriction of the bronchioles and attacks of dyspnoea. In its initial stages it is associated with corpulence and disordered digestion, and latter with congestion and swelling of the mucosa of the bronchioles, emphysema, and dilatation of the right side of the heart.

Pathology. Asthma is generally attributed to spasm of the bronchial muscles (Williams), and though recent observations have failed to sustain this, it must be admitted that in the majority of cases it is of decided neurotic origin. Again it is attributed to erythematous swelling in patches of the bronchial mucosa, (Clark). Another theory is that it is a fibrinous inflammation of the mucosa of the bronchioles, the tenacious exudate blocking the tubes more or less completely and relief coming with a more diffuent secretion. Berkart found a streptococcus in the sputa which he supposed to be the final cause. Again it has been held to depend on the circulation in the blood of deleterious matters, introduced during digestion. Again it has been attributed to a neurosis roused by constipation and the accumulation of irritant matters in the intestine. Whatever local conditions may be operative, there can be no doubt that in dogs it is almost exclusively confined to those kept indoors, overfed, without exercise, plethoric and constipated. The disease seems to originate in, and persist by, nervous disorder propagated from the digestive organs. A change of diet or any disturbing cause may bring on a paroxysm.

Symptoms. The disease is one of pet dogs, kept in towns, deprived of exercise, fresh air, and of the opportunity to relieve the bowels at will, and gorged with highly spiced meats, and sweets at least three times a day. Sluggishness and obesity are marked characteristics of the dog when first attacked, though in the advanced stages the violence of the paroxysms and their frequent recurrence may have induced extreme emaciation.

The affection is usually ushered in by a cough, at first slight, but soon becoming frequent, hard and sonorous, as in the early stages of bronchitis. The cough becomes very troublesome and the breathing habitually labored, but at irregular intervals a paroxysm comes on which threatens death by suffocation. The dog stands or sits on his haunches with open mouth, pendent tongue and staring eyeballs, panting for breath, and has his condition aggravated by every change of position or other source of excitement. By the frequency and severity of the attacks may be estimated the danger to the patient.

An examination in the intervals of the attacks detects some disturbance of the digestive organs. The tense and distended condition of the abdomen usually manifests the existence of overloaded stomach and bowels, of indigestion, tympany and constipation. Piles are often present as a result of long continued costiveness. The skin is dry and unthrifty, and often in patches denuded of hair. The teeth are covered with tartar and the breath fœtid.

Retching is occasionally seen to occur during a violent access of coughing, but only a little glairy mucus is brought up.

The cough, hurried breathing, and paroxysms of dyspnoea become aggravated, the general health suffers largely, and death often ensues in a state of great weakness and emaciation.

On *dissection* of such cases the lesions of various old standing diseases of the heart, lungs, or abdominal organs have been met with at times, and such disorders have doubtless assisted in maintaining and aggravating the asthma. The most constant lesions, however, are emphysema of the lung, and accumulations of fat in the mediastinum.

Treatment. 1st. During a paroxysm. This is confined to the administration of antispasmodics either by inhalation or as an injection, to avoid the additional suffering of swallowing.

Ether or chloroform may be inhaled from the sponge, but the employment of these should be guarded especially in advanced cases when, besides the prevailing weakness, there is reason to suspect structural changes in the heart. The same agents in doses of one, two or three teaspoonfuls, or laudanum in double that quantity may be thrown up as an enema, and may be combined with a couple of ounces of castor oil when costiveness exists. The fumes from burning stramonium or from burning brown paper which has been previously soaked in a strong solution of nitrate of potass, will in many cases suddenly cut short the paroxysm. If on the other hand there is reason to believe that the stomach is overloaded, the attacks will often be suddenly cut short by giving an emetic. For this purpose a grain of tartar emetic may be shaken upon the tongue, or a desert spoonful of wine of ipecacuan, or of antimony, poured down the throat. 2d. **In the intervals between the paroxysms.** Attention must be given to counteract any inflammatory action in the chest by which the disease may be maintained. Our chief object, however, must be to divest the animal of its superfluous fat and bring the digestive organs into a healthy condition. Unfortunately the propensity to fatten in some dogs seems to be a morbid condition. The food appears to be stored up as fat at the expense of muscular and other tissues even when the animal is kept on the borders of starvation. All flesh must be withheld and coarse vegetable fare alone allowed. A well boiled pudding (porridge) made with oatmeal or Indian corn meal, water and a little salt, with a small quantity of whey, skimmed milk or buttermilk, is an excellent diet in such cases. The amount must be small, though the hitherto pampered favorite will rarely seek to fully replenish his stomach until he has forgotten his former extravagant habits.

A good deal of open air exercise must be given, not violent, but gentle and long continued, and this though the patient may appear physically unfit for it. Exercise should be given three hours or more after a full meal. Purgatives (one ounce castor oil) should be administered twice a week. A clean bed, not too soft nor luxurious, should be allowed in a dry, airy place. The skin should be well brushed daily and occasionally washed thoroughly with soap, care being taken to dry the coat completely afterwards. Sedatives should be given daily, such as a half grain

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each of stramonium and tartar emetic, and in advanced stages with weakness and emaciation, vegetable tonics will be demanded.

Blaine strongly advocates a course of emetics, given every alternate day, and Mayhew lauds frequently repeated ammoniacal blisters to the sides. Such measures will be especially applicable when there is irritation and discharge from the bronchial mucous membrane. Only strong subjects can, however, bear such treatment.

All cases of asthma in the dog are obstinate and critical and require much judgment in treatment.

ASTHMA IN THE HORSE.

Hering records a case of *spasmodic asthma*, in a strong cart horse. Besides the oppressed and difficult respiration, the animal was excessively dull and had no appetite, but the pulse was almost of the natural standard. The animal was not benefited by opening medicine but improved under full doses of extract of hyoscyamus. Quillaume reports two asses attacked apparently in the same way, and Delwart and Robertson refer to other cases. They recovered under antispasmodics. These cases are at least closely related to heaves, which is largely a neurosis at first.

ASTHMA. BROKEN WIND. HEAVES. DYSPNŒA. PULMONARY EMPHYSEMA.

Definition. neurotic affection with digestive and respiratory disorders. **Causes :** no racial exemption, disease largely co-extensive with leafy hay from clover, alfalfa and other leguminous plants, musty hay, cryptograms, overloading the stomach, active work on a full stomach, overdriving, bronchitis. chronic bronchitis, emphysema. **Nature :** a neurotic affection, starting with derangement of some part of the vagus, dilatation of the right heart, congestion of the bronchioles. **Symptoms:** double expiratory action, flatulence, weak, husky, wheezing cough, glairy, grayish nasal discharge, wheezing, increased resonance along the margins of the lungs, sibilant râle, heart's impulse strong, even felt on right side, aggravation with overloaded stomach, costiveness or muggy atmosphere, improvement on laxative (green) food. **Treatment :** succulent green food, natural pastures, relieve any abnormal state of lungs or bowels, pure air, heart tonic, diet, arsenic, special diagnosis, guard against masking of symptoms by narcotics, privation of water, shot, lard, recto-vaginal fistula, diagnostic signs, dilated nostrils, auscultation and percussion signs of emphysema, relapse under hay and water.

Definition. A chronic affection of the equine species, manifested by a hurried, wheezy breathing, greatly aggravated by close, muggy weather, a full stomach, certain kinds of diet, or by exercise ; by a double lifting of the flank with each expiration ; by a small, weak, dry cough, often occurring in paroxysms and easily excited by a drink of cold water, exposure to cold air or a fibrous quality of the food ; lastly, by a marked disorder of the digestive organs.

Causes. This disease is essentially the result of faulty feeding and working, though age, pre-existing diseases of the air passages and sudden violent muscular efforts no doubt occasionally contribute to its development.

It has been alleged that some races of horses are exempt from this disease. Among these the Arabian, Persian, Barb, Spanish and Portuguese are especially named, but their immunity in all probability depends on the feeding and management rather than on any peculiarity of breed. The countries where these horses are met with are not subject to a prolonged winter but yield green food throughout the greater part of the year, and it is a notorious fact that no horse becomes broken-winded at pasture. The Arabians

moreover "feed their horses on the scanty plants which the borders of the deserts supply and when these are wanting they are fed on a little barley with chopped straw, withered herbs, roots dragged from the sands, dates when these can be obtained, and in cases of need, the milk of the camel. They drink at long intervals and in moderate quantities," (Low). Since an habitually overloaded stomach is the most common cause of *heaves*, the absence of the affection in the Arab is not surprising. But the Arab unfortunately enjoys no such security in England or America. Concerning the Barb, Delwart remarks that after a day's hard work, fasting, he is fed on six or seven pounds only of barley, and without the cut straw that the Arab is allowed in similar circumstances.

In Spain and Portugal horses at work are fed on broken wheat and barley straw, from twelve to twenty-five pounds, and barley from six to twelve pounds daily, according to the size of the animal and the demands upon his strength. The mares are constantly at pasture and according to the rainfall are starved or have abundance. Green food and a limited straw and grain diet are precisely the conditions in which *heaves* does not appear. Rodriguez, veterinarian to the queen, says that the disease was unknown to Spain until the cultivation of red clover, lucerne and sainfoin. At Aranjuez, horses fed on the hay of these plants, lost vigor and wind and several became decidedly broken-winded, All were, however, restored to health and vigor by a return to their former diet. Count Cardenas found that his horses gained in flesh on the new fodder, but that symptoms of *heaves* developed rapidly.

In France, M. Demoussy records similar facts. In Segala, where the aliment is substantially hay, broken-winded horses abound, whilst in the adjacent district of Causse where horses are fed through the winter on straw and barley, broken by the mule's feet in the act of threshing or treading out, the disease is virtually unknown.

Lucerne and sweet trefoil are indigenous and grow abundantly in Causse and Caussergne but eaten green or after their seed has been shaken off and the stems have acquired a dry ligneous character these are innocuous. When, however, condemned to stand in the stable through a severe winter with their racks constantly

filled with hay, horses will eat from thirty to thirty-five pounds of this daily and may become brokenwinded. The breeding mares which get little hay, seldom become affected, though the plentitude of their abdomen and the impaired respiratory function might be thought to conduce to the affection.

The leafy leguminous plants are slow to dry and as hay are always charged with cryptogams and bacteria and their products, which are manifestly a potent factor in causing heaves.

In England broken wind is much less prevalent than on the European Continent and it is deserving of notice that lucerne and sainfoin hold no place among the British green crops, that red clover hay is only exceptionally met with owing to the amount of land that is *clover-sick*, that natural hay is largely used, and that when horses are largely fed on hay it is qualified by such laxative agents as turnips, carrots, beets, etc.

All this throws light on the immunity of horses on our western prairies and plains. Feeding on the indigenous grasses, fresh or made into hay, they are saved from the noxious influence of those artificial products which are found, in all countries, to determine the development of heaves. It needs not that we adopt the popular notion that any special plant growing in these pastures ensures the safety of the equine races. It is merely a repetition in the Western Hemisphere of the experience so long before obtained in the case of Spain. Parallel with the progress of cultivation in our western lands, we see this malady advancing. Sixty years ago it was virtually unknown in Michigan and adjacent states, whereas now, these states can almost emulate New York in the relative number of their victims. It must not, however, be supposed that this cultivated fodder is the sole cause of the westward march of this malady. With improved agriculture have come better roads, spring wagons, and driving at a pace which was comparatively unknown to the early settlers.

In California, the condition of Spain was for long pretty accurately repeated. With no winter worthy of the name, troops of horses were left at pasture throughout the whole year and those that were stabled subsisted chiefly on natural hay in which the indigenous grasses were commingled with white—but no red—clover. California long retained the reputation of having no broken-winded horses.

In our Eastern states where the disease was thirty years ago so notoriously prevalent, the fields of luxuriant red clover might well have excited the envy of the English farmer. The hay made from this, full of seed, microbes, and toxins, was given without stint to the farm horses, which during the rigor of the winter were often shut up in stable for a length of time, and continuously and dangerously gorged themselves with this provender. In the Eastern States with a steady falling off in the red clover, there is also a corresponding reduction in the number of cases of heaves. The grain allowed them, a mixture, supposed to consist of Indian corn, oats and buckwheat, given as a dry, coarse flour, was little calculated to counteract the effects of the clover hay, and the entire absence of turnips and other succulent roots as a farm crop, precluded their use as a preventive of the malady. We need not forget the prevalent ambition to possess a fast trotter, nor the effect of the climate on the air-passages (See *chronic bronchitis*) in estimating the causes of this malady in the Eastern States.

The mere overloading of the stomach is a potent cause of the development of heaves. The horse is, above all other animals, compelled to undergo hard work on a full stomach. Coleman cites the experience of the coaching days when each horse had 20 lbs. of oats daily and not more than 5 lbs. of hay with no water before work. These horses were driven fast for long stages, yet they never contracted heaves under this treatment. Farmers' and millers' horses, on the other hand, were most subject to the disease because gorged continually with hay chaff and mealy food, and worked in this condition. "Nimrod", who confirms Coleman's statement says, "I have taken some pains to ascertain this fact by my own personal inquiries. One proprietor who has nearly fifty horses at work—many of which are in as fast coaches as any that travel on the road—assured me lately that he had not a broken-winded horse in his yard; whereas before he stinted them in their hay he generally had one to five in that state." Percivall testifies to its comparative infrequency in the English cavalry horses, which have their diet carefully regulated. Hay, musty from bad harvesting or other cause and such as is rank from growing in low wet localities is *caeteris paribus* more injurious than good hay.

Every day observation shows that driving a horse upon a full

stomach often causes heaves and nothing will more surely aggravate it, when it does exist. The same remark may be made of the drinking of large quantities of water after feeding and just before going to work. Gross feeders are above all others the subjects of the complaint.

The question arises how a disturbing cause, operating directly upon the digestive organs, should affect the respiratory, in such a marked and permanent manner. It cannot be because of the gastric and abdominal distension, since pregnant mares, though in a state of much greater plentitude, are not thereby rendered liable to heaves, and if they have previously suffered from this infirmity, the symptoms are usually less marked when breeding. The explanation first advanced by Dupuy appears to be the correct one. The lungs, the stomach, and certain other organs derive innervation from the vagus nerve, and certain disturbances of the stomach and intestines so impair the function of this nerve that the lungs are affected, at first functionally and afterwards structurally. In support of this view is the fact that heaves is usually associated quite as much with digestive as respiratory derangement. The horse though a heavy feeder becomes unthrifty, hidebound and emaciated; his dung is passed in an undigested state like so much chopped straw, and flatus is continually passed from the bowels. Indeed the almost incessant passage of wind and fæces, during the first mile or two of a journey, is a disgusting evidence of the malady. The power of doses of shot, fat and other agents to temporarily allay the symptoms may be held to point in the same direction.

Besides causes which operate primarily on the side of the digestive organs, others undoubtedly superinduce the disease, and among these severe exertions and chronic bronchitis ought to hold prominent positions.

Over-exertion induces over-distension and rupture of the air cells by the forced retention of air within the lungs, by the closure of the glottis, while the chest is strongly compressed by the respiratory muscles. It is an essential condition to all severe exertion in man that the breath should be held, and though the horse appears equal to the same efforts of draught after the operation of tracheotomy has deprived him of the power of holding the breath, yet he would seem to be sooner exhausted (Goubaux, Colin,

Bouley), from which it may be inferred that this power is frequently exercised, and it probably always is, in any sudden severe effort as in starting a heavy load, or jumping a five-bar gate.

This retension of air in the lungs during violent compression of the chest walls is precisely the condition met with during an access of coughing, and in both cases alike there is the tendency to overdistension of the minute tubes and air cells until they have lost their power of contraction, or they may even give way and allow the air to pass out and lodge in the lung tissue.

Another mode in which violent effort injures the lungs is by the rapid and continued inhalation of great quantities of air during rapid breathing, so as to dilate the lungs suddenly to their fullest extent. Sometimes from irregular distribution of the ærial current or from the want of tone in a particular part of the lung, it gives way under the pressure and the air cells become overdistended or ruptured. This condition is especially met in the more rapid paces. It is well exemplified in the results of the deep breathing after cutting the vagi nerves.

In either case the result will be more certain if the effort is made upon a full stomach, or with the functions of the vagus nerve impaired by a previous faulty diet.

That heaves is a frequent concomitant or sequel of *chronic bronchitis* is undeniable, and theoretically nothing is more likely to cause dilatation and rupture of the air cells and consequent impairment of the innervation and contractility of the lung than violent fits of coughing while the bronchial tubes have thickened and friable walls, or are partially plugged by tenacious mucus.

Heaves is mainly a disease of old horses, though I have seen several cases in five-year-old animals, and Bouley records a case in a two-year-old colt out of a badly broken-winded mare. This would seem to indicate a hereditary proclivity, and there is no doubt that the shallow, narrow, weak chest, predisposing to this as to many other pulmonary complaints, is transmitted from parent to offspring.

Leduc found in bad cases of heaves an unusual ossification of the costal cartilages, most marked at the chondro-costal joints where they were porous and red. The joint is often completely ankylosed and the slight normal movement in the tissues abolished. Even the chondro-sternal joints were stiffened, and

showed yellow spots, and commencing calcification of the articular cartilage. This inevitably interferes with the free mobility and dilatation of the chest wall, but the question remains whether these dystrophies, occurring as they do in the advanced stages of the disease, are primarily the cause of the impaired breathing, more than they are the result. Age even in sound horses brings a measure of calcifications of the cartilages, and excited breathing (overwork) must contribute to the same result. But if, as there is good reason to believe, acute cases of heaves in young animals are primarily induced by nervous (vagus) disorder, while the calcifying sets in later, it is begging the question to affirm that the as yet undeveloped ossification or calcification is the causative pathogenic factor. A surgical section of the chondrocostal joint can at best give but a transient relief of a morbid action, while the primal cause of the trouble remains untouched.

Nature of the Disease. Emphysema of the lungs is the most constant structural change met with in the bodies of animals which have suffered from heaves. This condition of the horse's lung appears to have been noticed by the early Greek writers. It was advanced as the cause of broken wind by Riding in 1704 (*Pathologie Veterinaire*), by Floyer in England in 1761 (*Treatise on Asthma in Man*), by Vitet in France in 1783 (*Medicine Veterinaire*, Lyon), by Frentzel in Germany, and Bracy Clark in England in 1795. It was only, however, after the admirable discoveries of Laennec that the question was systematically investigated by Delafond, who has furnished the most comprehensive data on the subject. Out of fifty-four broken-winded horses dissected by him no less than forty-five had the lungs extensively emphysematous. This emphysematous lung differs according to whether the emphysema is *vesicular* or *interlobular*.

In **vesicular emphysema** the smallest bronchial tubes and the air cells have become distended beyond the natural standard and remain permanently so, the lung tissue having lost its power of contraction. If such a lung is inflated and dried, and a thin slice taken from the surface of the emphysematous part, the size of the minute orifices on the cut surface will show its condition. These fine openings are only the air cells cut across, and in their healthy state they will admit no object larger than the point of a

needle or fine bristle. They are slightly larger in adult and especially in old horses than they are in the young. If affected by emphysema they will often admit a hempseed or even a small pea. On opening a healthy chest the lung collapses, contracting on itself and expelling the contained air; if the lung is emphysematous the diseased portion does not collapse, and if the entire lung is affected it continues to fill the chest and may even bulge outward after it has been opened. The color of the emphysematous lung is of a brighter red than are the healthy portions. If a diseased lung has been left exposed to the air for twenty-four to thirty-six hours and then cut across in all directions, the diseased lobules may be distinguished at a glance by this lighter shade, and such light portions, if near the surface, will be found to correspond to elevations above the general level of the lung. If the diseased lung is placed in water it floats on the surface like an inflated bladder, scarcely at all sinking into the fluid. If the lung is blown full of air the emphysematous part is first filled, causing the bulging on the surface to be still more marked than before. Vesicular emphysema rarely affects an entire lung; it is usually confined to the anterior lobes and to the thin lower and posterior borders of the organ.

Interlobular emphysema is the extravasation of air into the connective tissue between the lobules, owing to rupture of the air cells and smaller bronchial tubes. It may occur independently of the vesicular emphysema but more frequently it is preceded by that form and results from it. It is manifested on the surface of the lung by irregularly formed transparent elevations movable from one place to another under the pressure of the finger, contrary to what is the case in vesicular emphysema. These vary from the size of a pea to that of a hen's egg. When the air exists in the cellular tissue between the lobules it appears as intersecting lines, circumscribing irregular spaces, and seemingly dark colored to a superficial glance but seen to be transparent on a closer examination. Like the elevations on the surface these collapse on being pricked.

When a lung in this state has been inflated and dried it presents on the diseased parts the union of several air sacs into one by the rupture of their intervening walls, so that a pea may be lodged in the cavity; it further shows wide and prolonged canals

on the surface and in the intervals between the lobules—the dilated areolæ of the connective tissue. These abnormal conditions like the vesicular emphysema are chiefly met with in the anterior lobes of the lungs, along their free borders and on their inner surface near the entrance of the bronchi.

One or both of these two forms of emphysema may be considered as essential conditions in all forms of heaves. It does not follow that this is the primary disease; we have already seen that the cause of the malady is usually to be sought on the side of the digestive organs, and that impaired innervation, on the part of the vagus nerve or of the ganglia in the brain presiding over it, leads to these functional and structural changes in the lungs. If these changes are results and not causes, their extent will not necessarily bear a constant proportion to the intensity of the disease, though in reality they are generally found to do so.

From a series of injections of lungs from broken-winded horses, M. Demoussy arrived at the conclusion that the essential lesion of broken wind was an aneurismal dilatation of the capillary vessels of the lung. This is like the condition of the mucosa found in asthma in man and is explainable in both cases by the impaired innervation, as dilatation of these minute vessels is a natural consequence of the loss of vaso-motor nervous power, and contact with air saturated with carbon dioxide.

Dilatation of the smaller bronchial tubes is frequently present and especially characterizes such cases as supervene on chronic bronchitis. These dilated tubes contain a plastic, whitish inodorous mucus.

Another frequent concomitant of the emphysematous lung is a dilatation of the right cavities of the heart, especially the auricle, and an attenuation of their walls. The same condition is noticed in pulmonary emphysema in man and like this is probably due to the slow and imperfect circulation in the diseased lung.

Collating these structural changes with the different causes of the disease, we find that they harmonize with the theory of impaired function on the part of the vagus nerve or its presiding ganglia, whether this functional disturbance has its origin in disorder of the digestive organs, as in the great majority of cases,



in the cryptogams and microbes in the leafy leguminous hay, in severe muscular efforts, or in chronic bronchitis.

Section of the vagi nerves affords an exaggerated instance of their paralysis and its results. These are mainly emphysema, capillary dilatation, blood extravasation, inflammation and pulmonary collapse. Emphysema is the first result and due to the slow, deep respiration (Boddaert) and loss of contractibility (Longet); capillary dilatation results from the extreme distention of the air cells and the retention in them of air, highly charged with carbon dioxide (Donders); the other lesions occur later and own very different causes.

That this is the true nature of the disease would further appear from the occurrence of emphysema without heaves, two cases of which are recorded by Percivall; and from the existence of broken wind without emphysema. Cases of this last variety have been recorded by Godine, Volpi, Rodet, D'Arboval, and Delafond in France; and by Sewall, Dick, Smith, Hallen and Gloag in Britain. In connection with this last class of cases, it must be noted that dilatation of the right cavities of the heart sometimes gives rise to very similar symptoms, and that the signs of chronic bronchitis are often scarcely distinguishable from those of heaves. In catarrhal bronchitis, too, after the air tubes have been washed, it is sometimes impossible to decide whether the lining membrane has been the subject of inflammation or not. For anchylosis and calcic cartilages, see above.

Symptoms. The most prominent are the double lift of the flank with each expiratory act in the absence of fever, the short, weak, dry and almost inaudible cough, the wheezing noise in breathing when that is accelerated by exertion, and the intestinal flatulence with the frequent passage of gas.

The cough usually heralds the advent of other symptoms. Often the character of the cough draws forth the remark that an animal is becoming broken winded and though no other symptoms are seen at this time, they thereafter rapidly develop. At this early stage of the disease, the cough is paroxysmal, coming on in fits during work or after a drink of cold water. Once the disease is established the horse rarely coughs more than once at a time. The cough is extremely short, weak and low and followed by a sort of wheeze. So specific is it that if once

heard it can readily be recognized. The sudden effort made in coughing usually leads to the expulsion of gas from the flatulent bowels.

The double lifting of the flank in expiration is not peculiar to broken wind. It is seen as well in most diseases of the lungs and even of other organs (enteritis, peritonitis), which interfere with the freedom of the respiratory act. If, however, it is not attended by fever but associated with the broken-winded cough, the wheezing respiration, the disordered and flatulent state of the bowels, the tumultuous beating of the heart against the left side after exertion, and the slight flow of clear watery matter from the nose, it is pathognomonic. The act of inspiration is quick and free, that of expiration is not uniform and continuous as in health, but consists of two stages interrupted by a momentary arrest. In the first stage the posterior part of the abdomen is slightly raised and it falls in laterally; then comes an almost imperceptible period of inaction followed at once by the further lifting of the flanks to complete the expulsion of air from the lungs. The first stage seems the natural collapse of the walls of the chest and forward movement of the diaphragm, the second a contraction of the abdominal muscles partly due to the exercise of will to overcome the obstacle to the expulsion of air.

In very bad or advanced cases these symptoms are more marked. The inspiration is sudden and manifested by a rapid expansion of the chest, and dropping of the belly, previously supported by active contraction of the abdominal muscles. The two stages of the expiratory act are quite distinct. The first is manifested by a sudden falling in of the walls of the chest so that the ribs no longer stand out prominently beyond their interspaces; the abdomen equally rises inferiorly and falls in laterally so that a projecting ridge is formed from the lower end of the last rib to the point of the hip. This is especially marked during the period of inaction, and this is succeeded by the second effort, quick and almost convulsive. These movements are so extensive that they are conveyed in a striking degree to any vehicle to which the animal is attached, especially if it has only two wheels, and a rider on horseback feels the movement still more disagreeably. When a horse is in this state the alternate rising and falling of the abdominal organs imparts a synchronous movement of pro-

trusion and retraction to the anus and in thin subjects a rising and falling of the muscles on each side of the root of the tail. The nostrils, too, are kept constantly dilated.

There is a *nasal discharge*, but this is very inconsiderable in the early stages of the malady. It is a clear watery or slightly grayish, albuminous material without any visible admixture of pus globules, and on drying, it leaves a scarcely perceptible crust. At first it appears intermittently, and in minute quantities, but in bad cases it becomes almost constant, and is especially profuse after exercise.

Abnormal respiratory sounds are marked symptoms in the advanced stages. The wheezing noise of the breathing, especially when that is excited by exertion, may be heard at a short distance from the animal. The increased resonance on percussion along the lower border of the lung is only heard when the emphysema is extensive. The dry sibilant or whistling r  le heard over the same parts is equally a symptom of the advanced stages. When there is much discharge a moist rattle is often heard over the lower end of the windpipe or immediately behind the middle of the shoulder. The overlaying of the anterior lobe by the the thick muscular shoulder, and the complication of symptoms obtained at the free border of the lung by reason of the abdominal noises and resonance, render these results less conclusive in the earlier stages and slighter cases.

The application of the hand to the side of the chest behind the left elbow may detect a strong impulse of the heart with each beat. If the patient is actively exercised for some time this may be felt on the right side as well. This symptom indicates the existence of dilatation of the right cavities of the heart.

The symptoms of indigestion are also very manifest. The dung passed is like so much chopped hay and oats, and does not at all resemble the f  ces of a healthy horse. The abdomen is tumid, tense and filled with flatus, which is frequently passed *per-ano*, and has no doubt given rise to the name of **broken wind**. This expulsion of gas from the rectum usually takes place whenever the animal is excited to cough. When first started on a journey, the frequent passage of wind and dung for the first mile or two is one of the most disagreeable features of the disease. When the animal has thus *emptied himself* he usually goes much better for the remainder of the journey.

Broken-winded horses are always greedy feeders, and if they get little work they manage to maintain their flesh. But they are soft and flabby, and if put to active work they fall off rapidly, becoming emaciated and hidebound, a true indication of their impaired digestion.

The symptoms are liable to occasional aggravation. If the stomach and bowels are overloaded they are invariably so. If the patient is kept in a hot, close stable, the same result follows. Thick, muggy weather has the same effect. After a more than usually severe day's work all the symptoms may be intensified, and this may continue for several days or a week. Bouley attributes this to an extensive rupture of air cells and a sudden increase of emphysema, and the gradual subsidence of the symptoms to the partial absorption of the displaced air and the accommodation of the lung to its new condition.

Light and laxative diet, on the other hand, alleviates the symptoms and a broken-winded horse usually improves at grass.

Richter and Schmidt note that when the victim of heaves has its temperature raised from 37.7° to 40° C. by work, the hyperthermia goes down very slowly under rest, so that it is still abnormally high three hours later; whereas a healthy horse having its temperature similarly raised by work, may have it reduced over one degree by 15 minutes of rest, and to the normal in two hours. As a diagnostic measure this is too tardy, and it becomes uncertain in all cases in which violent exercise develops a congestion which will lengthen the period of hyperthermia though no heaves may be present.

Course. The general tendency of heaves is to persistent aggravation, but by a judicious regimen many cases may be checked in their progress and greatly relieved, or even cured.

Treatment. We have already seen that heaves is virtually unknown on natural pastures where the grass is short, green and succulent. Turning out on such pastures will improve or even temporarily cure mild cases. The same may be said of the laxative systems of diet. (See that recommended for *chronic bronchitis*). Feeding on dry grain only, with a very limited supply of water, will enable many broken-winded horses to do ordinary work with comparative ease and comfort. In such cases, however, improvement is only due to the empty and unclogged

condition of the digestive organs and the symptoms return with all their former intensity when the original diet is restored. By way of palliation much may be secured by avoiding accidental causes of aggravation. If catarrh or bronchitis has supervened, it should be treated in the ordinary way. If the stomach and bowels are overloaded and costive, a small dose of aloes and enemata will relieve. If the stable is close a free admission of air will be beneficial. The temporary excitement in these cases may be further alleviated by sedatives, of which opium and digitalis have been mostly employed. The last agent will sometimes control the breathing to such an extent that the horse may be thought to have completely recovered. Professor Dick believed that he had effected a cure in one case by the administration of a single dose of a drachm each of camphor, opium, calomel and digitalis. Tincture of lobelia $\frac{1}{2}$ oz., extract of grindelia robusta 1 oz., or kerosene oil 5 ozs., will abate the symptoms. Temporary results only can, however, be expected from such agents, except in the case of an aggravation due to a cause acting for a limited time only, in which case the partial improvement may be lasting.

By adopting such measures to check accidental complications and confining the animal to a rigid system of diet, a broken-winded horse may be worked with comfort to himself and his master. The aliment should be principally or exclusively of oats, bran or barley, though good succulent grass, turnips, carrots, beets, and potatoes may be allowed, as may also wheat or oat straw in limited quantity, but no hay and, above all, none prepared from red clover, alfalfa, sainfoin, or allied forage plants, and none that is musty or otherwise injured by keeping. No feed nor water must be allowed for one or two hours before going to work, and the pace must be slow at first and gradually increased as the horse empties himself, and the breathing gets less embarrassed. If meadow hay, straw or other bulky food is allowed in small quantity this must be after the horse has returned from his day's work.

If the food above recommended is boiled or pulped, and mixed with some saccharine agent as molasses, its restorative action is enhanced.

If, however, we add to these hygienic and dietetic measures a

prolonged course of arsenic, the symptoms generally disappear. From five to seven grains of arsenic made into a powder with a drachm of bicarbonate of soda and ten grains of digitalis may be given daily in the food until improvement is noticed or symptoms of the poisonous action of the agent appear. When these are manifested in loss of appetite, colicky pains or red and watery eyes, the medicine must be suspended and begun again some days later in smaller doses.

The therapeutic value of arsenic in this case is probably largely due to its action on the nervous system, which has long been recognized. As early as the first century of the Christian Era, Dioscorides recommended its use in asthma, and in recent times it has acquired considerable reputation for the treatment of neuralgia. Another—though perhaps an allied—physiological action of arsenic no doubt adds to its value in this equine disorder. This is its power of retarding the waste of tissues. This property it possesses in common with tea, coffee and some other agents, but to a greater degree. This has led to its extensive employment by the peasants in Lower Austria, Styria, and the mountains separating Austria from Hungary, who found that it improved their personal appearance, increased their weight and enabled them to sustain greater exertions in climbing without fatigue. It was the revelations of Dr. Tschudi concerning the Styrian arsenic eaters that first led Professor Bouley to try this agent in heaves.

Examination of Broken-winded Horses. Though the symptoms enumerated above are sufficient to detect heaves in all ordinary cases, yet it may not be time thrown away to caution the reader against pronouncing it absent when the more prominent symptoms are not seen. Unscrupulous dealers do not hesitate to avail of a variety of devices to conceal the symptoms and make the animal pass for a sound horse. Digitalis and other sedatives are so employed, but are mostly rejected because they render the horse dull and sluggish. By some the bowels are unloaded by a dose of physic, the horse is kept on a spare diet of oats, beans and other grain, water is withheld, and on the morning of sale one or two pounds of leaden shot or of bacon fat are administered. The inconvenience attendant on the presence of these agents in the stomach makes the animal desist as much as

possible from moving the abdominal organs, and the double lifting of the flank is thus more or less completely hidden. With the veterinarian, however, this measure, like the last, defeats its own purpose, for such horses are always intolerably thirsty and if allowed to regale themselves at the nearest watering trough, the charm is broken, the double lift returns and with it all the symptoms of the malady.

A brutal practice existed among ancient farriers, of making an artificial opening into the rectum to allow the exit of the flatus upon which they conceived the disease to depend. This was effected either by cutting through the sphincter ani with a knife or by making a new opening to one side of it with a red hot iron. According to Ferguson this has been improved upon by the modern Irish jockey, in the case of broken-winded mares. With the knife an artificial communication is made between the rectum and the vagina, of sufficient size to insure that it will remain open and large enough to allow pellets of dung to pass into the vagina. The double lifting of the flank forces the fæces through this artificial opening, and to avoid the inconvenience of their presence in the vagina the animal carefully refrains from this action. This orifice further allows the free escape of any gases generated in the rectum and thus materially relieves the flatulence. Ferguson says he has seen broken-winded mares that have been operated on in this manner, that breathed so freely that even professional men have failed to detect the affection.

In all cases of broken wind, no matter how masked, there will be manifest, on slight exertion, a permanent dilatation of the nostrils—*i. e.*, alike in inspiration and expiration,—and when any such suspicious symptom is seen the horse should be carefully examined, especially the state of his lungs, as ascertained by auscultation and percussion, his breathing after he has freely partaken of water and hay, and, if there is suspicion of drugging, after he has stood over night in a hot stable, plentifully supplied with both hay and water.

It should be borne in mind that mares advanced in pregnancy often show no double action of the flank though decidedly broken-winded.

POLYPUS OF THE BRONCHIAL TUBES.

Like other mucous membranes, that covering the bronchial tubes is liable to diseased growths, which may each remain attached by a broad base, and form a morbid elevation of the surface, or it may become loosened and retain its connection with the mucous membrane only by a neck or pedicle. An interesting case of the latter variety is recorded in the *Edinburgh Veterinary Review* for January, 1864, by Mr. Parker of Birmingham. The polypus was attached to the wall of the right bronchus about an inch below the bifurcation of the trachea, and had an ovoid form measuring $8\frac{1}{2}$ inches in its longer circumference by $4\frac{1}{4}$ in its shorter. The pedicle was $1\frac{1}{2}$ inch long and allowed the tumor to pass freely upward into the lower part of the windpipe, threatening instant suffocation. It had a fibrous struture and was continuous with the interlacing bundles of yellow elastic tissue which cover the cartilaginous rings of the bronchus.

From its frequent displacement upward the tumor gave rise to paroxysms of hurried and difficult breathing, apparently threatening suffocation, but when these passed off, respiration was tolerably tranquil and easy. The fits of dyspnœa came on after any unwonted excitement, but above all after a cough. In such cases the tumor had been coughed up into the lower end of the windpipe, and until it slid back into its former position, the animal seemed on the very verge of death. The paroxysms had appeared very frequently during a period of five months that had elapsed since her purchase, increasing steadily in severity and finally causing death. The lungs contained many small abscesses the result doubtless of the frequent paroxysms of dyspnœa.

Cases of this kind are not usually amenable to treatment, nevertheless as they are pretty certain to end fatally if neglected, it would be quite permissible to perform tracheotomy in the lower part of the neck and attempt to snare the tumor with an elastic wire passed through a single or double tube. The tumor might even be seized by a vulsella and twisted off provided it could be reached.

NEOPLASMS. TUMORS OF THE LUNG AND PLEURA.

While tumors belong rather to surgery than medicine, yet when present in the lung, they are as a rule inoperable and come directly under the care of the physician who has almost exclusive charge of this organ.

Fibroma. In the horse, Kitt mentions a single case of fibromata, in size from a nut to a closed fist, encircling the root of a lobe, so as to cause strangulation and collapse of the lung. They were connected with the pleura and bronchia. In a case recorded by Blanc, they grew from the phrenic pleura.

Fibrous neoplasms and nodules are common in all domestic quadrupeds in connection with persistent inflammation (see Chronic Fibrous Pneumonia.)

Sarcoma. Simple sarcoma is common in the lungs of horses, cattle and dogs; in *horse* scattered through the lung tissue, (Cadeac, Bruck, Perini,) or on the pleural surface (Adam, Dieckerhoff); in *cattle* in lung tissue and pleura, (Leger, Hemminger, Morot); in *dog*, in the lung and pleura, (Cadeac). The lungs are very commonly involved in these small-celled fleshy tumors when they have become generalized elsewhere.

Melanoma. Pigmentary Sarcoma. These are very common in *gray horses* in which melanosis has become generalized. In a case at N. Y. S. Veterinary College, lungs and pleuræ were plentifully sprinkled with melanotic deposits, but beside this almost every organ in the body was involved. Among gray horses, turning white or already white, these tumors are well nigh universal and when generalized the lungs are usually involved. Among other colors they are uncommon.

In *cattle* melanosis is very rare. Gurlt, Hertwig and Fiorentini notice black patches on the surface of the lungs in newborn calves but these disappear as the animals grow. In *dogs*, Lafosse describes pigmentary deposits in the form of black lines, but pigmentation may be held to be the normal condition of the surface of part of the canine lung and there need be no apprehension of this developing into a tumor. Brückmüller and Cadeac however record genuine pigment tumors of the lungs in connection with melanosis of the lips.

Carcinoma. These are common in the lung and even in the

pleura of the *horse*, but usually in connection with generalized carcinoma (Langeron, Bean, Hutyra, Gramlich, etc.) Baillet mentions a case in which was a 7 lbs. mass, lying in the division of the bronchi, and leading to the compression of vessels and nerves in that vicinity. In *cattle*, Lucet cites cases in the bronchial glands and mediastinum. In *dogs*, Kitt notes the existence of carcinomata, of a size of from a millet seed to a pea scattered along the bronchial mucosa, and obstructing the bronchioles. This was in a generalized case. Kitt notes a case in the *cat*.

Endothelioma. Miliary neoplasms of the pleural epithelium in the *horse* are recorded by Morot, extending even into the lung substance. Hutyra quotes a case in the *dog*, in which the neoplasms took the form of papillomata two thirds of an inch long.

Epithelioma. These have been found in two cases in the lung of the *horse* (Nocard, Schütz), and more frequently upon the pleura and pericardium but also in the bronchial and thyroid glands (Schindelka, Weber, Barrie). They form in clusters which project from pulmonic and parietal pleuræ like tubercles. In *cattle* they have been found in the lung, generally in connection with an epithelioma of the larynx (Kolb), and in *sheep* in the lung (Besnoit.) They were small hard miliary granules which could easily be felt by passing the hand over the surface of the lung. In *dogs* they are found in the substance of the lung and on pleura and pericardium, sometimes along with similar formations in the peritoneum (Montané, Cadeac, Lienaux).

Adenoma, Lymphadenoma. *Lymphadenoma* has been found in the *horse's* lung in the form of small nodules (Laulanié, Montané), and *Adenoma* in the papillary form in cattle (Eber).

Chondroma. One case in the lung of a *horse* is reported by Schmidt, the diseased organ weighing 61 lbs. It is described as a chondro-adenoma. Other cases affected the costal pleura. In *cattle*, Weltzer records a case of chondroma growing on the cartilage of the bronchus and completely blocking the passage. It was very solid and hard, and covered by a fibrous sac. In the *dog*, Nocard and Cadeac narrate the presence, in different cases, of multiple chondroma of the lung, a single tumor in one case weighing 11 lbs. and extending through the diaphragm into the

abdomen. On section it showed a yellow, nearly homogeneous surface, with hæmorrhagic areas.

Osteoma. Bony tumors have been especially noticed in the lungs of *cattle*, the new formation having the resistance and consistency of cancellated osseous tissue (Coremans). This may be associated with the manifest tendency of tubercles to calcification in these animals, and the ossification of the bronchial rings in calves as noted by Kitt. In *dogs*, Cadeac records as a frequent occurrence, the formation on the surface of the lung and even throughout its substance of fine calcified granules, giving a characteristic roughness to the pleural surface. They are densely white and each surrounded by a thin zone of fibrous tissue. Their cause is unknown, but probably they are connected with parasitism or bacterial infection.

Lipoma. Fatty tumors have been found in the *dog's* lung, in some cases generally disseminated through its substance (Semmer).

Symptoms of Pulmonary Tumors. These are generally in the nature of interference with respiration in general, varying in different cases according to the seat and bulk or the multiple condition of the neoplasm, and again as to the nature and abundance of the discharge. There is usually no attendant fever, and the trouble has been slowly progressive rather than by sudden access. It may be, and especially in the early stages, that no symptom is shown when at rest, but the breathing is quickly disturbed by active exertion. Under such circumstances, or in advanced stages, with extensive or numerous tumors, there may be accelerated difficult often catching breathing, dilated nostrils, and even projecting eyeballs, a deep, painful cough occurring in paroxysms, a loud ronchus if the bronchia are compressed or partially filled, and a flatness on percussion in the seat of any large tumor. In such a situation the normal respiratory murmur is absent, and there may be blowing sounds, mucous râles, heart sounds, rumbling or gurgling from the transmission through the solid body of sounds of distant organs. There may also be areas of drumlike resonance on percussion and wheezing, in connection with the development of emphysema. Nasal discharge may be absent, or if the bronchia are involved it may be abundant, and of various kinds. In melanosis there is more or less black liquid in which the pigment granules or masses are clearly seen under

the microscope. In carcinoma the discharge may be bloody and under the microscope, multinucleated and giant cells may prove suggestive. In advanced carcinoma there is liable to be anæmia, pallor of the mucosa and a sunken condition of the eyes.

In cattle there is in addition, irregular or suspended rumination and tympany. In all animals emaciation appears early and progresses steadily.

No *treatment* of the tumor of the lung is satisfactory. The invasion of the lung may sometimes be prevented by the early excision of tumors of a primary nature, appearing externally.

PARASITES OF THE LUNGS, HEART AND PLEURA.

<i>Parasite.</i>	<i>Habitat.</i>	<i>Host.</i>
<i>Aspergillus fumigatus</i> ,	Lungs,	Horse, Ox, Birds, Man.
<i>Actinomyces</i> ,	Lungs, Pleura,	Ox, Horse, Man, etc.
<i>Cytodites nudus</i> ,	Air sacs,	Gallinacæ.
<i>Linguatula denticulatum</i> ,	Lungs, Bron. glands,	Herbivora.
<i>Distoma magnum</i> ,	Lungs,	Cattle.
<i>Monostoma mutabile</i> ,	Bronchia,	Goose.
<i>Paragonimus Westermanni</i> ,	Lungs,	Man, Dog, Cat, Pig.
<i>Bilharzia crassa</i> ,	Blood,	Ox.
<i>Cysticercus Tenuicollis</i> ,	Pleura,	Ox, Sheep, Goat, Pig.
<i>C. Bailleti</i> ,	Pleura,	Dog, Cat.
<i>Echinococcus</i> ,	Pleura, Lung,	Ox, Sheep, Goat, Pig, Horse, Man.
<i>Strongylus Filaria</i> ,	Bronchia,	Sheep, Goat, Camel, Dromedary, Deer, Argali, Gazelle.
<i>S. Rufescens</i> ,	Bronchia, Air cells,	Sheep.
<i>S. Capillaris</i> ,	Bronchia, Air cells,	Sheep, Goat, Roebuck.
<i>S. Micrurus</i> ,	Bronchia,	Calves, Cattle.
<i>S. Pulmonaris</i> ,	Bronchia,	Calves, Cattle,
<i>S. Arnfieldi</i> ,	Bronchia,	Horse, Ass, Mule.
<i>S. Elongatus (Paradoxus)</i> ,	Bronchia,	Swine.
<i>S. Pusillus</i> ,	Bronchia,	Cat.
<i>S. Commutatus</i> ,	Bronchia,	Hare, Rabbit.
<i>S. Vasorum</i> ,	Heart, Pulmonary Vessels, Bronchia,	Dog.
<i>S. Subulatus</i> ,	Venous blood,	Dog.
<i>Sclerostoma equinum</i> ,	Blood vessels, Lungs,	Horse, Ass, Mule.
<i>Spiroptera Sanguinolenta</i> ,	Lungs,	Dog.
<i>Filaria Immitis</i> ,	Heart, Pulmonary artery, Pleura,	Dog.
<i>F. Sanguinis equi</i> ,	Blood,	Horse.
<i>Syngamus trachealis</i> ,	Trachea, Bronchia,	Gallinacæ, Turkeys.
<i>S. Bronchialis</i> ,	Trachea, Bronchia,	Duck, Swan, Goose.
<i>Balbiana gigantea</i> ,	Heart,	Ruminants, Pigs.

DISEASES OF THE MEDIASTINUM.

Anatomical Conditions. The mediastinum is the central space in the chest, limited on each side (right and left) by the two layers of parietal pleuræ, which close the cavities (pleural) occupied by the right and left lung respectively.

It contains the heart and pericardium, occupying, in the horse, the space between the third and seventh ribs, and above this the roots of the large vessels (aorta, anterior and posterior aortas, pulmonary artery and its first divisions, the anterior and posterior venæ cavae), the vena asygos, the thoracic duct, the trachea with its bifurcation, the tracheal and bronchial lymph glands, the œsophagus, and the vagus, phrenic, sympathetic and left recurrent laryngeal nerves. In front of the heart it contains the two innominate arteries and their divisions, the confluence of the jugular veins, and anterior vena cava, and their affluents, the trachea and œsophagus and their lymph glands, the remnant of the thymus, and the vagus, sympathetic, phrenic and left recurrent laryngeal nerves. Behind the heart the structures occupying the mediastinum are the œsophagus and œsophagean lymph glands, the abdominal branches of the vagus, sympathetic and phrenic nerves, and at its upper border the posterior aorta, vena asygos, thoracic duct, sympathetic nerve trunks and ganglia, and the dorsal lymph glands. Smaller lymph glands and nodes are scattered throughout the mediastinum, and into the lymph glands drain the lymph currents from the structures adjacent to each (lung, bronchia, trachea, œsophagus, heart, pericardium, pleuræ, thymus, supporting connective tissue, nerves, vessels, etc.) Thus any advancing or infective disease in any of these organs is liable to produce secondary affections in the mediastinal lymph glands. Their intimate connection with the diaphragm and through that and the thoracic duct with the abdominal organs, lays these glands open to infection and disease which has commenced outside the thoracic cavity proper.

INFLAMMATION OF THE MEDIASTINUM : MEDIASTITIS.

This may be acute or chronic, simple or infectious, primary or secondary. It is usually concentrated in the lymph glands, yet it may extend around these, or, starting independently in the lymph plexuses, it may lead to extensive exudation and infiltration of the connective tissue, with subsequent organization and contraction which compresses important organs and impairs vital functions. This may compress the heart, interfering with its normal functions, but much more frequently it acts through obstruction of the vena cava, with consequent tardy movement through the jugulars and capillary congestion of the neck and head or of the fore limb; or it lessens the calibre of the pulmonary veins, threatening congestion of the lungs and causing dyspnoea; or again it obstructs the aorta or pulmonary artery, causing irritability and irregularity of the action of the heart, with hypertrophy or dilatation; or again it compresses the vagus or sympathetic nerve, producing hepatic, gastric or intestinal disorder; or compressing the left recurrent laryngeal nerve it leads to paralysis and wasting of the arytenoid muscles of the larynx, with stridor (roaring) in forcible inspiration; or it presses on the gullet, interfering with deglutition and eructation, and producing indigestion and tympanies.

In the more acute cases in which the phlegmonous inflammation eventuates in abscess, this may become enclosed in thick, fibrous walls as a permanent source of dyspnoea, or more commonly, it breaks into the pleural cavity, producing *empyema*; or it may break through the foramen dextrum or sinistrum into the abdominal cavity with fatal results; or, more happily, it may burrow toward a bronchial tube and discharge through that or between the two first ribs so as to escape at the lower part of the neck.

In horses such acute cases are usually manifestations of *irregular strangles*, but they may also supervene on pneumonia, croupous or contagious, on infectious bronchitis, on pleurisy, on pericarditis, on influenza, or even on other pyogenic infection. The chronic cases are more likely to coincide with glanders, tubercle, actinomycosis, hydatids, linguatula, or neoplasms—simple or malignant.

In **cattle**. Acute cases may result from perforation of the gullet in ill directed interferences in cases of thoracic choking, or again in acute inflammations of the lungs (lung plague, contagious pneumonia, inhalation broncho-pneumonia), or in pleurisy. The more tardy cases are likely to follow on neoplasms, tuberculosis, hydatids (echinococcus, cysticercus tenuicollis) or actinomycosis.

In **sheep**, in addition to choking, pulmonary inflammation and tuberculosis, there are parasitisms (lung worms, hydatids, linguatula) and infectious caseous adenitis.

In **dogs** the affection may attend on distemper, infectious bronchitis, ossifying enchondroma (Nocard), and other neoplasms, simple and malignant.

In **dogs, cats and pigs** lacerations and other injuries of the gullet with sharp-pointed bones became a cause of mediastinal inflammation, especially adenitis.

Symptoms. In acute cases supervening on other inflammatory diseases of the chest the symptoms are masked by the previous and persisting signs of surrounding disease, and as objective physical signs are obscure in the larger quadrupeds, the case is not discovered until the necropsy. When not covered up by such concurrent symptoms, there is usually marked dyspnœa, tenderness perhaps on percussion on the median line of the sternum, difficulty in swallowing, in cattle absence of eructation and the supervention of gastric tympany, in dogs and pigs nausea and vomiting, and in some cases paresis of the larynx and stertor.

When the connective tissue is thickened, organized and pressing on the tissues, another class of symptoms may be added: There is tense dilatation of the jugulars persisting even during inspiration, the result of obstruction of the blood-flow in the anterior cava; there may be œdema of the lips, nostrils and eyelids, dark red congestion of the conjunctiva and nasal mucosa, and fulness of the cervical and facial veins. The dropsy may extend to the fore limbs and even to the hind. In the human subject the suppression of the pulsation during inspiration to reappear with expiration is a marked feature. There may follow hepatic congestion, dropsy and ascites. In such a case the veins of the trunk, hind limbs, and fore are prominently dilated, as well as those of the neck and head. The heart sounds are dull and

muffled owing to the compression of the base of the heart and the interference with its systole.

In case of abscess advancing forward, a doughy, painful swelling may show in the right or left jugular furrow. A previous chill may have suggested the formation of abscess, and a similar occurrence with spreading tenderness over the chest or abdomen may indicate the escape of pus into one of these cavities. As cases of this kind in solipeds are usually irregular forms of strangles, the earlier symptoms of this affection throw light on the true nature of the later lesion.

Treatment is eminently unsatisfactory. It must be directed to control and limit the primary disease and thus ward off the more dangerous sequel. In strangles the securing of an early maturation and free discharge of the intermaxillary or pharyngeal abscess will do much to ward off such complications as we are considering. When mediastinitis has set in, it may be treated like pleurisy or pericarditis, but less hopefully; when empyema or suppurative pericarditis is discovered, drainage and antisepsis will be in order. When an abscess points at the lower part of the jugular furrow we must favor its maturation, open as early as possible and treat the sac antiseptically, packing with bismuth paste.

When the mediastinal exudate has become organized and presses on important organs nothing can arrest its progress nor relieve the organs from pressure.

NEOPLASMS OF THE MEDIASTINUM.

The respiratory, nervous, circulatory and digestive disorders that result from inflammation of the mediastinum are still more common as the result of new growths in this situation.

Fibroid degeneration (sclerosis) of the lymph glands may be set down as the simplest of such conditions. Under the influence of some irritant, often ptomaines or toxins from a diseased lung, bronchium, pericardium or pleura, the lymph glands become congested and gradually hypertrophied. At first of a more or less vivid red, they become gradually pale, firm, and hard, the fibrous elements of the gland increasing in thickness, and the inflammatory exudate becoming organized into fibrous tissue, thereby causing compression and even atrophy of the gland tissue. When the afferent trunks bring not only toxic

matters but also pus cocci, numbers of small abscesses, miliary or larger, are formed through the parenchyma. In exceptional cases an abscess may attain to the size of a hen's egg and remain as a cold abscess, or burrow toward the pleura, pericardium, bronchus, peritoneum or breast.

From such enlarged glands a series of symptoms may arise like those resulting from simple inflammation of the mediastinal connective tissue, or organization of the inflammatory exudate.

The *treatment* of such cases is not hopeful unless the original cause of irritation can be put a stop to, when in recent cases the persistent use of iodide of potassium may reduce the glandular hypertrophy. In old standing sclerosis of the glands no such happy issue can be expected.

Lymphadenoma may occur in connection with leucocytosis and produce all the evil results of compression enumerated above, The symptoms would resemble those already mentioned and the diagnosis would be completed by the excess of leucocytes in the blood, and often by the pallor of the visible mucosæ and swelling of superficial lymph glands. In this case attention should be given to hygiene, open air life, generous feeding and iron and other tonics to build up the strength and increase the red globules.

Melanoma. In solipeds (the gray and white especially) and less frequently in other genera, pigmentary deposits are found in the mediastinal lymph glands, as in other parts, and as they may grow to any size and show no limit as to numbers, the results are the same as in the other forms of compression above described, Along with the general symptoms, the presence of melanomata elsewhere and, in horses, the white or gray color will serve to identify the melanotic formations.

Sarcoma has been found in the œsophagean glands in cattle (Bournay), **papilloma** on the pillars of the diaphragm (Pradal), and **cystoma** at the root of the bronchi (Adenot) in the same genus, and **carcinoma** is common to domestic animals generally.

Ossifying Chondroma has been found by Nocard in the mediastinum of the dog, and indeed any form or malignant neoplasm may be expected in this situation with a manifestation of more or less of the symptoms found in productive inflammations. These may, one and all, be pronounced to be beyond reach of successful treatment.

ACTINOMYCOSIS OF THE MEDIASTINUM.

Cattle are especially subject to the invasion of the tissues by the star-fungus, and since once implanted, it progresses in entire disregard of the nature of the tissue in its way, it invades the mediastinum, connective tissue, glands, etc., and may grow to a large size and exercise most injurious compression on the structures involved or adjacent. It is most likely to make its entrance from the gullet or bronchi, but may also reach the glands through the lymph channels from lung, pleura or pericardium. If its presence can be reasonably inferred from the symptoms, and the concurrence of similar formations near the surface, it may be treated, and usually successfully, by the free administration of iodide of potassium, 16 grains to every 100 lbs. of live weight, daily for four or five days, then leave an interval of two days, giving a laxative, then another course, and so on for a length of time.

TUBERCLE OF THE MEDIASTINAL GLANDS.

As tubercle is essentially or præeminently a disease of the lymphatic system, and is concentrated in the lymph glands that receive the drainage from tuberculous tissues, the bronchial and mediastinal glands become involved in nearly all tubercular affections of the bronchia, lungs and pleura, and to a less extent in similar affections of the diaphragm and abdominal organs. So notorious is this, that, in any case of suspicion of tuberculosis of these parts, we make a point, in a necropsy, of first examining the glands in the mediastinum as the most likely to throw light on the more obscure lesions of the tissues. It is no uncommon thing to find the lymph glands in the mediastinum tubercular, when no indication of the disease is found in the lungs, pleura, pericardium or diaphragm. The bacilli have passed through these tissues without causing any lesion, or the slight lesions which they caused have healed, without leaving any appreciable, permanent, structural change.

Cattle and swine, as being most frequently affected with tuberculosis, show the mediastinal lesions most commonly, but all animals suffering from thoracic tubercle usually show the lesions in the glands.

The lesions may be slight, consisting in small, pea-like nodules often with caseated or calcified centres, or the whole gland or group of glands may be similarly invaded and necrotic, making a diseased mass of 12 inches in its longest diameter. There may or may not be infiltration into the surrounding connective tissue. In such cases the pressure upon vital thoracic organs may be such as to produce the train of symptoms mentioned above under *mediastinitis*. In cattle, constant tympany of the rumen is a prominent feature, becoming aggravated after meals. The normal healthy eructations of gas may be suspended, dyspnoea appears under slight exertion and distension of the jugular, cervical and facial veins are often marked symptoms. When the tubercular glands have attained a great size, the dull, flat sound on forcible percussion may show their outline. In any case a cough is easily roused by exertion, and if the lungs are not seriously implicated this is likely to be especially dry or husky. Other signs of tuberculosis may be shown: in the lungs circumscribed areas of flatness on light percussion and of wheezing or other abnormal sounds, with, in some instances emphysematous, drumlike, tympanitic percussion sound; in the throat, wheezing, guttural breathing and distinct swelling; in the abdomen capricious appetite, impaired rumination, a tendency to scour on forced feeding, and enlarged mesenteric glands felt through the rectum; in the superficial lymph glands asymmetrical enlargement, etc. In all cases of suspicion the tuberculin test should be applied.

While some cases survive slight tubercular infection and even recover, there is no such hope in connection with extensive deposits, and, as in fatal, chronic infectious diseases generally, treatment only becomes a means of perpetuating and extending the infection, thus giving permanence and prevalence to a disease which should be completely *stamped out*. If preserved at all, for breeding purposes, it should only be in the most rigidly secluded herds from which all opportunity for extension is carefully excluded. (See Tuberculosis Vol. IV),

GLANDER NODULES IN THE MEDIASTINUM.

Like tuberculosis, glanders is a disease of the lymphatic system especially, and in case of a localized glanderous deposit, the lymph glands which drain the lymph from that point are constantly involved. Therefore in pulmonary glanders, the bronchial, tracheal and even the mediastinal glands become involved. As in tubercle the centre of the gland lobule is especially implicated, its cell elements undergoing degenerative necrotic processes with more or less liquefaction or caseation. The afferent lymph vessels are liable to a marked hyperplasia causing these to stand out in abnormally firm, rigid cords. Yet the nodules do not habitually attain to the large size so frequently seen in tuberculosis, so that the compression of the surrounding organs, and their functional derangement are less marked. There is, however, the chronic cough, with more or less dyspnoea under exertion, a slight grayish, sometimes sticky nasal discharge, and often distinct glanderous deposits at other points. The presence of the characteristic ulcers in the nose, the nodules or cicatrices on the mucosa, the cording of the lymph vessels running toward the submaxillary lymph glands, the comparatively insensible, hard nodular outline of these glands, the presence of circumscribed swellings with corded lymphatics, nodules and open sores on a limb or on the trunk, together with a knowledge of a present or previous existence of glanders in the locality, or of an exposure to the infection, would furnish ground for grave suspicion. The mallein test is the final and crucial resort in all suspicious cases.

As in tuberculosis, and even more so, the preservation and treatment of cases of glanders is essentially subversive of sound sanitary administration, and is an infallible indication of ineffective and comparatively useless sanitary work. Mild cases will long survive the disease and even recover, but they do far more harm by the spread of the infection than can be gained pecuniarily from their preservation. (See Glanders, Vol. IV).

CASEATING LYMPH-ADENITIS OF SHEEP.

Sheep are subject to an infectious disease of the lymph-glands resulting in a caseous degeneration of the gland tissue, which was formerly confounded with tubercle.

History. The first case on record was seen in 1891 by Preisz and Guinard. Other cases were recorded by Preisz in 1894. Turski in 1897 recorded 44 cases in 150 sheep in West Prussia, all from the same estate. In these the bronchial and mediastinal glands were especially involved though not to the exclusion of the portal, inguinal and precucular glands. In no case were lungs or liver affected. Later, other cases were found in the Gotha (Saxony) abattoirs (Ostertag). In 1899 Cherry and Bull recorded cases observed at Melbourne, Australia, the prevalence amounting to from 15 to 70 per cent. in certain flocks. In these the following glands were affected: prescapular, superficial inguinal less frequently, the scrotal, pelvic and mediastinal. Once the kidney was diseased, but the mesenteric glands or liver never. In 1899, Savori records the disease as affecting 10 per cent. of the sheep killed in the abattoirs of Buenos Ayres. The bronchial and mediastinal glands were mainly affected, and caseous bronchopneumonia and adhesive pleurisy were common, as were also caseous mesenteric glands, and nodules in the liver and kidneys.

In the United States it was first noted in Southern California in 1897, and immediately after, in Chicago, South Omaha and Kansas City (Norgaard and Mohler). Out of 16,000,000 sheep killed at these points 3236, (.02 per cent,) were condemned as affected with this disease. The disease has been less noticed in the Eastern States yet it must be accepted as widely diffused in all quarters of the world.

Age. In all cases the old sheep suffered more numerously than the young. At Los Angeles, Cal., but one lamb suffered in 3,500, while among 756 wethers 50 were affected, and in 194 ewes 23 were diseased. The ewes not only suffered in greater numbers but showed more extensive lesions than the wethers. Though all over 6 years old, and therefore discarded from breeding, these ewes were not disqualified for good fattening and slaughter. Age therefore and long and repeated exposure to the

infection must have favored extension of the disease, while the prime of life and strong vigorous health proved no bar to infection. Indeed the disease has been seen mainly in fat sheep killed for mutton.

Causes. Bacteriology. The essential cause of this disease is a short, stubby, round ended, ovoid microörganism. It is non-motile, ærobic (facultative anærobic), non-sporulating, stains readily by Ziehl's, Löffler's and Gram's methods, is very susceptible to the action of acids. It shows a remarkable polymorphism. It may be present as a coccus, a diplococcus (dumb-bell), or a club-shaped organism, the latter 1.3 to 1.6 μ long, by 0.4 μ thick. Though this growth is at first tardy in artificial culture, it proliferates much more freely in successive generations, on agar, glycerine agar, peptonized beef buillon, or blood serum. It grows poorly on gelatine, as it does best at 37.5° C. which melts the gelatine medium. It grows freely on potato and in milk. It is not gas producing with sugars. The thermal death point is 65° C. for 10 minutes. It resists frost, but is killed by a 2.5 per cent. solution of carbolic acid for 1 minute, or by a 0.25 per cent. solution of formalin for 6 minutes, or a 1:2000 solution of mercuric chloride for 4 minutes.

Exposure to limewater for 24 hours did not appreciably retard subsequent growth, suggestive that the germ may be preserved in limestone soils or such as are rich in organic matter.

Animals susceptible. Feeding experiments on Guinea-pigs caused death in from five to eight weeks with caseous degeneration of the lymph glands. Inoculation of rabbits caused death in from three to seven weeks, and feeding experiments in from eight to ten weeks. Caseation of the lymph glands resulted in both cases. Mice, pigeons and hens succumbed much more slowly. Caseation of lymph glands followed intravenous and subcutaneous inoculation in sheep, but not after feeding experiments.

Symptoms. The disease is usually overlooked during life because of the absence of definite symptoms. Nearly all the cases studied have been discovered *post mortem*, the sheep having been killed in good condition for mutton, and without any suspicion of illness. Inoculated cases have been found to fall off in condition and weight for two or three months, and the fact that

casual cases have been mostly in old ewes rejected as being no longer useful in the breeding flock, and put up to fatten, argues a visible loss of health and stamina which led their owner to turn them into mutton. Yet this did not prevent improvement under liberal feeding and the animals came to the knife in good flesh. When superficial lymph glands are affected (prescapular, pharyngeal, precrural, inguinal) more definite symptoms are found, the glands being enlarged to the size of a hen's egg or even a closed fist, perhaps giving a sensation of cheesy contents, and causing stiffness of the limbs. Under a scrutinizing observation of the flock the extensive lesions of internal organs could not be overlooked. Hurried breathing, dyspnoea and cough under exercise, would point to deposits in the lungs or mediastinal glands; yellowness of the mucosæ, impaired appetite and rumination might point to disease of the liver; frequent urination and stiffness and dragging of the hind quarters might imply disease of the kidney. In a contaminated flock, or on infected land a steady loss of condition on the part of the mature animals only, might well suggest the presence of this disease.

Morbid Anatomy. The caseated centres are usually seated in the lymph glands, but they are not always confined to these. They may be found along the line of the pastern, metacarpus or metatarsus, or elsewhere in the connective tissues. Nørgaard and Mohler figure a deposit on the upper surface of the sternum in a deep pit excavated in the bone, and I have found a similar deposit on the floor of the spinal canal, and quite frequently around the pharynx in pampered Cotswold sheep. The glands that are especially obnoxious to attack are the bronchial, mediastinal, prescapular, precrural, superficial inguinal, scrotal, sublumbar, deep inguinal, pharyngeal, and less frequently the suprasternal and mesenteric.

When recently invaded the gland is red, swollen and congested but still firm. Its cut surface exudes a watery fluid. In this there appear later several whitish centres of degeneration and softening, which gradually increase in size and finally coalesce into one common caseous mass, enclosed in a thick resistant capsule. The contents have at first the consistency of thick cream, which may become inspissated so as to resemble rich, old, ripe cheese, and finally a dry, floury powder. These caseous con-

tents are remarkable for their yellowish green color, which recalls the intestinal nodules caused by the *Æsophagostoma Columbiana*. The capsule forms a smooth, clearly defined limitation membrane, separating the mass from the surrounding healthy tissues. When caseation has taken place there is no external zone of congestion nor inflammation as in tuberculosis or glanders,

In the early nodules before caseation, the tissue is seen to consist mainly of leucocytes and nucleated cells, round, or of an irregular outline and granular. In the centre of the nodule the cells are already breaking up and disintegrating into a granular debris. The bacteria, oval dumb-bell or club shaped, are found among and even within the leucocytes and mingled with the debris.

The miliary nodule in the liver of an inoculated animal, killed after three weeks, is thus described by Norgaard and Mohler :—
“ A caseous centre composed of an amorphous material which does not take any of the ordinary stains. Surrounding the centre may be seen numerous leucocytes, more or less degenerated and frequently containing one or more bacilli, while clumps of these organisms are scattered among them. External to this is a dense round-cell infiltration, the peripheral zone of which is undergoing connective tissue metamorphosis and serving as a line of demarcation between the atrophied liver cells and the central cell-mass. This process then repeats itself centrifugally, causing the appearance of concentric layers, until a sufficiently strong reaction takes place to form a connective tissue barrier strong enough to encapsulate the central part of the nodule and prevent its further growth.”

Before the mass is encapsulated in this way the bacteria easily escape into the lymph channels and are washed on to form the nuclei of new nodules in the lymph glands and even in the lymph plexuses of connective tissue. In this way may be explained much of the work of generalization in which multiple nodules and centres of caseation are found in the lungs, bronchial walls, liver, spleen, kidneys and other parts.

Differential diagnosis. From phlegmonous abscesses this is to be distinguished by its slow progress, by the absence of acute febrile symptoms, and of the common pyogenic germs, micrococci, staphylococci, streptococci, bacilli, and by the presence of

the polymorphous, ovoid, dumb-bell or club-shaped bacterium of Preisz. From tuberculosis and glanders it is differentiated by the absence of the delicate, slow-staining, rod-shaped bacilli of these diseases, and the presence of the bacterium of Preisz. The clear, smooth, homogeneous surface of the fully formed capsule in the caseous gland, also serves to distinguish it from the irregular surface of the tubercle or glander nodule with its evidence of peripheral congestion and advancement into surrounding tissue, and the coincidence of lesions of old and recent date. Again tuberculosis may be identified by the tuberculin test, and glanders by the mallein test. From the nodular disease of the intestines (*Æsophagostoma*) which it resembles in its yellowish green color, it is clearly distinguished by its seat of election, preferring as it does the lymph glands, and avoiding the intestinal walls and mesentery.

Treatment. In the case of superficial caseous deposits, a free incision followed by an injection of zinc sulphate $\frac{1}{2}$ oz., carbolic acid 1 dram, and water 1 quart, repeated daily, until the cavity heals by granulation, serves to repair the local lesions and to reduce the number of caseating centres which are undermining the general health.

Prevention. This does not seem to have been attempted mainly, perhaps, owing to the fact that the disease is not often fatal, and that the affected sheep can be fattened and marketed. Yet the prevalence of the affection in given flocks and pastures, while others are spared, suggests its restriction by yearly, or at least frequent changes of pasturage, the old pastures being plowed up and subjected to a rotation of cultivated crops. Water that has drained from the contaminated soils must be avoided, and buildings and yards and, above all, racks and troughs that have been used by affected sheep must be disinfected. In introducing fresh blood into a flock, or on to a pasture hitherto free from the disease, care should be taken to avoid infected flocks and pastures as the source of supply.

PARASITES OF THE MEDIASTINUM.

The parasites of the mediastinum will be more fully described in volume V, but it seems appropriate here to at least name the different species and their hosts :

<i>Parasite.</i>	<i>Habitat.</i>	<i>Host.</i>
<i>Linguatula Denticulata</i> , (Larva of <i>L. Tænioides</i>)	Lymph glands,	Sheep, goat, antelope, deer, dromedary, ox, horse, Guinea-pig, cat, etc.
<i>Symplectoptes cysticola</i> ,	Connective tissue	Gallinaceæ.
<i>Echinococcus Veterinorum</i> ,	Connective tissue, Heart,	Man, domestic animals.
<i>Cysticercus tenuicollis</i> ,	Connective tis- sue, serosæ,	Ruminants, pig, cat, man.
<i>C. Pisiformis</i> ,	Connective tis- sue, serosæ,	Rabbit, bird.
<i>Cœnurus Serialis</i> ,	Connective tissue,	Rabbit, hare (goat).
<i>Bilharzia crassa</i> ,	Blood,	Ox, (Egypt).
<i>Paragonimus Westermanni</i> ,	Bronchia,	Dog, pig, cat, man.
<i>Spiroptera Sanguinolenta</i> ,	Œsophagus,	Dog.
<i>S. Megastoma</i> ,	Œsophagus, mucosa,	Soliped.
<i>S. microstoma</i> ,	Œsophagus,	Soliped.
<i>S. (Gongylonema,) scutata</i> ,	Œsophagus,	Soliped, ox, sheep, pig
<i>S. Uncinata</i> ,	Gullet,	Goose.
<i>Filaria immitis</i> ,	Heart, blood,	Dog.
<i>F. Osleri</i> ,	Bronchial tubercles,	Dog.
<i>F. irritans</i> ,	Blood,	Horse.
<i>F. Sanguinis, equi</i> ,	Blood,	Horse, (Egypt).
<i>F. Hæmorrhagica</i> ,	Blood,	Horse.
<i>F. Anatis</i> ,	Around heart,	Duck.
<i>Strongylus filaria</i> ,	Bronchia,	Sheep, goat, camel.
<i>S. rufescens</i> ,	Bronchia,	Sheep, goat, camel.
<i>S. micrurus</i> ,	Bronchia,	Calves.
<i>S. pulmonaris</i> ,	Bronchia,	Calves.
<i>S. Arnfieldi</i> ,	Bronchia,	Horse, ass.
<i>S. Capillaris</i> ,	Bronchia,	Goat.
<i>S. Elongatus</i> ,	Bronchia,	Pig.
<i>S. Pusillus</i> ,	Bronchia,	Cat.
<i>S. Commutatus</i> ,	Bronchia,	Rabbit, hare,

<i>S. Vasorum,</i>	Heart cavities,	Dog,
<i>S. Subulatus,</i>	Blood,	Dog,
<i>S. Nodularis,</i>	Gullet,	Duck, goose.
<i>Trichina Spiralis,</i>	Connective tissue,	Pig, man, etc.
<i>Sclerostoma syngamus,</i>	Bronchia,	Birds.
<i>S. Equinum,</i>	Aorta,	Soliped.
<i>Ollulanus tricuspis,</i>	Connective tissue,	Cat.
<i>Larva of Œstrus equi,</i>	Gullet,	Horse.
<i>Larva of Œstrus Hæmorrhoidalis,</i>	Gullet,	Soliped,
<i>Larva of Œstrus pecorum,</i>	Gullet,	Soliped.
<i>Larva of Œstrus duodenalis,</i>	Gullet,	Soliped.
<i>Larva of Œstrus bovis,</i>	Around gullet,	Ox.
<i>Aspergillus fumigatus,</i>	Bronchia,	Birds, mammals.
<i>Albiana gigantea,</i>	Œsophagean and cardiac muscles,	ox, goat, sheep, pig.

DISEASES OF THE HEART AND ORGAN OF CIRCULATION.

Susceptibility in different genera. Reasons for partial immunity of the quadruped. Special and general causes in quadrupeds: violent, forced work, fatty degeneration, swallowing of pointed metallic bodies, difficult diagnosis in the animal. Position of the heart in the horse, ox, sheep, pig, carnivora, birds. Structure of the heart as a pump. Results of imperfect structure or action. Heart-walls. Table of size of the heart. Capacity. Weight. Pulse in each healthy genus, according to age, size, environment, temperament, proximity to parturition. Morbid conditions of the pulse: frequent, slow, quick, tardy, full, strong, weak, feeble, indistinct, small, hard, wiry, thready, oppressed, leaping and receding, intermittent, unequal, irregular, anæmic, venous. Percussion. Palpitation. Auscultation. Healthy sounds. Morbid sounds, in unusual place, force, intensity, rhythm, repetition of 1st sound, of 2nd sound. Murmurs, synchronance with given stages of heart movement, their significance, pericardial murmur. General symptoms of heart disease, cold extremities, passive congestions, dropsies of limbs, etc., shortness of breath, venous pulse, vertigo, dullness, sluggishness, corpulence.

The lower animals are perhaps less subject to heart disease than mankind, but the comparative immunity generally assumed for them is far from being a real one. The horizontal position of the quadruped largely obviates that special tax upon the heart demanded by the erect position of man, and especially by the elevated place given to his more ample and vascular brain. Animals too are comparatively free from those mental and moral influences which so largely affect the regularity of the circulation in the human subject. But on the other hand many physical causes of heart disease affect the lower creation equally with their lord, while some undoubtedly operate with special force on the brute. All animals are subject to disease of the heart as of other internal organs, from exposure; this organ is occasionally involved from its contiguity with other diseased structures or from interdependence of function as we have already seen in certain diseases of the lungs (congestion, heaves, etc.); the tendency to heart disease frequently runs in a particular family of animals, especially with the rheumatic constitution, which is transmitted from parent to offspring as surely as the color of the skin the turn of the horn or the depth and spring of the rib. The lower animals

are further subject to congenital malformations and imperfections and to deposits of morbid material around the heart or in its substance so as to impair its healthy action.

Horses and dogs have special predisposing causes in the violent and prolonged exertion to which they are habitually exposed. The quiet sluggish and non-excitabie ox and pig meet with dangers no less real, though of a different kind, in the overfeeding which induces fatty degeneration of the heart as of other muscular tissues. The larger ruminants are further endangered by their propensity to swallow needles, wires and other sharp pointed bodies which ultimately reach and penetrate the heart.

The prevalence of heart disease in animals may be deduced from the fact that out of 150 horses, oxen and dogs dissected at Montfaucon by Leblanc in 1840, not less than one-twentieth presented cardiac lesions. The supposition of an immunity of the lower animals, has been largely due to the heavy muscular shoulder of quadrupeds which covers the upper and anterior regions of the heart, shutting them out from easy physical exploration. In man the entire heart and connecting blood vessels are so open to examination, that the physician can pronounce with the greatest accuracy, not only concerning the existence of disease, but also its precise locality and nature. In the quadruped no such facility is open to us, and veterinarians have too generally refused to face the difficulty, preferring to ignore heart diseases, or still worse seeking to cover their ignorance by the assertion that such affections rarely exist. Now, however, we not only know that heart diseases are much more frequent in the lower animals than heretofore believed, but that as a general rule they are sufficiently manifested and recognizable by their distinctive symptoms.

Position and exposure of the heart. In the horse the heart has only its apex and a small portion of its left ventricle approached to the surface of the chest, at a point where it is felt to beat behind the left elbow. The apex approaches the surface in the interval between the fifth and sixth ribs and close above the breast bone. The posterior border of the ventricle follows a nearly vertical line upwards from this point, while the anterior border has a direction upward and forward, crossing diagonally over the fifth rib. The part of the ventricle exposed extends about three

inches upwards from the apex, and is about two inches in its traverse diameter. The great mass of the organ is covered by lung substance.

In the **ox** about the same extent of heart tissue is exposed. In **sheep** a portion about an inch in height, and one and a half inches in breadth, is left uncovered by lung. In the **pig** the heart is exposed only in a triangular space of about an inch across.

In the **carnivora** the heart lies more directly in the median line of the chest. It appears as if tilted forward, so that its apex is directed backward, and its base forward, while the body of the organ lies directly over the breast bone. The lungs invest it on both sides preventing any approximation to the walls of the chest laterally, and it can best be auscultated by applying the ear over the sternum.

In **birds** the heart is situated in the center of the chest and enveloped by lung tissue so that its exploration is about equally difficult at all points.

The larger blood vessels at their origin from the heart are not open to examination in the lower animals, except to a limited extent in the dog.

Internal arrangement and structure of the heart. In all warm-blooded animals the heart is composed of two portions, the internal cavities of which, are perfectly distinct from each other, and contain blood in different conditions; the right portion holding the impure, purple or venous blood which has just circulated through the body, and the left portion being filled with the bright crimson or arterial blood, which has been aerated by circulating through the lungs. Each of these portions is divided into two distinct cavities, an *upper* (auricle) which receives the blood from the veins, and a *lower* (ventricle) which receives the blood from the auricle and transmits it into the arteries. The auricle is separated from the ventricle by a traverse musculo-membranous partition having a large central orifice furnished with valves (auriculo-ventricular), the free borders of which are turned downward, so that they allow the blood to flow freely downward from the auricle, but completely close the orifice and prevent any reflux when the ventricle contracts. The great artery which originates from the base of each ventricle is likewise furnished with a system of valves (semi-lunar) having their free borders turned into

the artery, so that they allow blood to flow freely into that vessel during the contraction of the ventricle, but prevent any reflux into the heart when the ventricle again dilates. The apparatus may be likened to a force pump with two systems of valves, one to prevent the return of any water from the pump into the fountain ; the other to hinder any reflux from the delivery pipe into the pump. Any interference with either of these valves entails a very serious and usually a fatal disorder of function.

These *orifices* differ considerably in size. Those between the auricles and ventricles are considerably larger than those at the commencement of the great arteries. Those on the right side of the heart too are greater than those on the left. They vary with the form of the heart. Thus in dilatation of an auricle and ventricle on one side of the heart, the auriculo-ventricular opening becomes equally widened, and the valves remaining disproportionately small, the blood is allowed to rush back into the auricle during ventricular contraction. The left auriculo-ventricular opening has been known to become contracted in some very flat and shallow chests ; the blood failing to circulate freely through the lungs and to reach the left side of the heart in a full supply, this orifice accommodates its size to the amount, and may become so narrowed that it forms a serious obstacle to the blood flow, and a series of morbid changes result, following a backward course the reverse of the circulation. The auricle first becomes overdistended, and its muscular walls increase in thickness and consistency ; the lungs tend next to suffer from a passive congestion, and lastly the right side of the heart becomes engorged and enlarged.

Any obstruction in the aorta which conveys the blood from the left side of the heart, equally leads to dilatation of its internal cavity and abnormal thickness of its walls.

The imperfection of the valves is one of the most serious results of such changes in heart structure. The sounds by which such imperfection may be recognized will be presently noticed, meanwhile the mode of testing this in the heart of the dead animal will be referred to. If due to structural changes in the valves themselves, the new deposits, the cicatrices, the lacerations, etc., will be visible to the eye. Though no such disease-changes are seen, the valves may still manifest imperfection by failing to ful-

fill their normal functions when put to the test. Water is poured into one or other of the great arteries which arise from the ventricles, the vessel being held vertically, and if it fails to descend into the heart the valvular action is perfect. The ariculo-ventricular valves may be equally tested by filling the ventricle and observing whether there is a reflux into the auricle.

The thickness of the walls of the heart varies in disease. The auricular walls are invariably thin and flaccid except as above noted with diminution of the ariculo-ventricular orifice. The walls of the right and left ventricles differ in thickness in accordance with the distance to which they have respectively to propel the blood and the propulsive effort demanded. Thus the walls of the *right ventricle*, which is only called upon to propel the blood through the lungs, are only about $\frac{1}{2}$ an inch in thickness and are thinnest at their lower part. Those of the *left ventricle*, which has to send the blood to the most distant parts of the body, are from 1 to $1\frac{1}{2}$ inch except at the lower part where they form the apex of the heart, and are reduced to a tenuity resembling the walls of the auricles. They are thickest at the medium part, and diminish slightly in an upward or downward direction. The bulk of these walls is excessively muscular, the fibres arranged as an elaborate double spiral and connected with a layer of white fibrous tissue placed in the interval between the auricles and ventricles, and surrounding the ariculo-ventricular openings and the orifices by which the great arteries take their origin. It is at this point, where the muscular fibres of the ventricles are connected with the white fibrous rings, where rupture of the heart usually takes place.

The following measurements may be held to refer to medium sized animals of the different kinds mentioned :

	Longitudinal Diameter. Inches.	Transverse Diameters at the Base of the Ventricles,		Circumference. Inches.
		Antero-Poste- rior. Inches.	Transverse. Inches.	
Horse -----	10	$7\frac{1}{2}$	$5\frac{1}{8}$	$19\frac{1}{2}$
Ox -----	$9\frac{4}{5}$	$6\frac{4}{5}$	$4\frac{1}{2}$	$17\frac{1}{2}$
Sheep -----	4	3	2	$7\frac{1}{2}$
Pig -----	$4\frac{1}{2}$	$3\frac{3}{5}$	$2\frac{1}{8}$	$9\frac{3}{5}$
Dog -----	$3\frac{2}{5}$	$3\frac{2}{5}$	2	$8\frac{1}{2}$

The internal capacity of the ventricles is so modified by the amount of *post mortem* contraction that it differs widely from the actual capacity during life. The left ventricle of the larger domestic quadrupeds usually admits from $3\frac{1}{2}$ oz. to over 5 oz., while the right ventricle with walls so much thinner and more lax will contain double that amount. In the smaller animals about a tenth of these quantities will be admitted.

The weight of the heart, too, can only be stated as an average or for medium sized animals. In the *horse* it may be from $4\frac{1}{2}$ lbs. to 9 lbs. ; in the *ox* from 3 lbs. 5 oz. to $4\frac{1}{2}$ lbs. ; in the *sheep* from $5\frac{1}{2}$ oz. to 7 oz. ; in the *pig* from $9\frac{1}{2}$ oz. to 14 oz. ; and in the *dog* from 5 oz. to 7 oz. This statement must be understood to apply to dogs approximating in size to the shepherd's.

Taking into account the size of the particular animal any considerable deviation from these measurements and weights may be accepted as abnormal. The ratio to the body weight is about :—horse and dog 1 : 100, ox, sheep and pig 1 : 220. This necessarily varies with condition—fat or lean.

The pulse offers valuable indications in disease of the heart.

The number of the pulse in healthy full grown animals may be set down as follows per minute :—horse, 36 to 49 ; ox, 38 to 42 (with loaded paunch or in a hot stable up to 70) ; sheep, goat and pig 70 to 80 ; dog, 80 to 100 ; cat, 120 to 140 ; goose, 110 ; pigeon, 136 ; chicken, 140. In old age the pulse is less frequent. This diminution may extend to 5 beats per minute in the larger quadrupeds and to 20 or even 30 in the smaller. Youth and small size are again associated with a greater rapidity of the pulse. The pulse of the foal, at birth, is about three times that of the horse ; in the colt of six months it is double ; at a year old about one and a half times ; and at two years old one and a quarter.

The smaller the animal, *cæteris paribus*, the more rapid is the pulse. Hot buildings, exertion, fear or any other exciting cause likewise accelerates it. It is more frequent with the nervous temperament, as for example in the English race horse, or the greyhound, than in the dull lymphatic cart-horse, or mastiff. In advanced pregnancy it is increased in number. In the cow and mare it undergoes a monthly increase of four or five beats per minute after the sixth month. (Delafond).

Independently of these conditions, a rapid pulse indicates febrile

excitement attendant on active inflammatory or other disease, or a state of weakness and debility. In this last condition the heart beats more frequently to secure a more rapid circulation in the capillary blood vessels, and thus make up to the craving tissues by frequency of contact, what is wanting in the quantity and quality of the nutritive fluid. This point cannot be too much insisted upon, as the fatal doctrine, that a rapid pulse indicates force of the circulation is very misleading as to treatment.

The force and character of the pulse differ in the various species. In the horse it is full, moderately tense and elastic. In the ass and mule it is smaller and harder, with an inequality of force in successive beats and sometimes even a beat is suppressed or imperceptible. In the ox the pulse is full, soft and regular, appearing to roll forward beneath the fingers. In the sheep and goat the pulse is small, but with a peculiar quick or sharp beat. The pig's pulse is said to be firm and hard. That of the dog and cat is firm and hard, coming with a sharp impulse against the finger. In the dog, however, successive beats are not always of the same force, and an intermission or complete absence of a beat is by no means an indication of disease of the heart or other serious malady. It often attends the slightest excitement in a perfectly healthy animal.

In disease the *pulsations* may become :—**frequent** or increased in number ; **slow** or decreased in number ; **quick** or striking with a sharp impulse against the finger ; **tardy** or without sharpness of stroke and as if they rolled slowly past under the finger ; **full** and **strong** when the impulse is forcible and not easily compressed by the finger ; **weak**, **feeble** or **indistinct** in the opposite conditions ; **small** when though perfectly distinct and forcible they are wanting in fullness ; **hard**, when forcible and jarring (this is sometimes called **wiry** or, if smaller, **thready**) ; **soft** when, though the artery may be full, the beat is devoid of hardness and easily compressible so as to be unfelt ; **oppressed** when with a full rounded artery, the impulse is jerking though not hard, and as if the distended vessels opposed the transmission of the impulse ; **jerking** and **receding—leaping**, when with empty and flaccid arteries the pulse seems to leap forward with each beat of the heart—(this pulsation may be visible to the eye in the carotids) ; **intermittent** when after a number of beats at regular in-

tervals there is a complete pause extending over that period of time which would have been occupied by a full beat ; **unequal** when some beats are strong and others weak ; **irregular** when without any distinct intermission for a period equal to that of a single beat, the intervals between successive beats are of varying length. The pulse further has a peculiar **thrill** or **tremor** in states of great debility with deficiency of blood and imperfect filling of the vessels.

Of these the **leaping**, the **intermittent**, the **unequal** and the **irregular** pulses are of special importance in their bearing on heart diseases.

The **jerking** and **receding** pulse is felt in cases of imperfection of the semi-lunar valves at the commencement of the great aorta, which allows blood propelled into the arteries by the contraction of the ventricle to flow back into the ventricle during its state of relaxation. This pulse is met with in other conditions as in aneurism of the aorta, but if from heart disease, it is distinguished by the presence of a *blowing murmur* with the second sound of the heart.

The **intermittent** pulse indicates functional derangement of the heart but it does not as is generally believed betoken structural disease. It is frequently observed in healthy asses and mules, and in dogs however slightly excited whether by fear or joy, or by the mere fact of their being handled, it is so common as to be almost the rule rather than the exception. It may be seen in a healthy horse as the result of excitement. During the early stages of convalescence from inflammatory affections of the lungs in the horse the pulse is often intermittent. The pulsations are at the same time unequal. There is a regular cycle of beats gradually decreasing in force and extending over a complete respiratory act. The cycle commences with the strongest beat during or immediately after the act of expiration, and the succeeding four or five beats are less and less forcible until the chest is fully expanded when there is a quiescent interval corresponding to the period of one beat. In many such cases there is no other indication of heart disease and the phenomenon appears due to the interference with the circulation by the hepatized lung, to the impaired nervous energy of the heart and to its compression between the distended lungs. A pulse simply *intermittent* and not assoc-

iated with any further sign of heart disease, does not then possess the significance generally attributed to it, but a careful examination of the heart should invariably be made when this functional disorder is observed. It exists or may be brought about by slight excitement in the great majority of heart diseases.

In case of intermittent pulse it is useful to ascertain whether there is also an intermission of the heart's beat, since in softening of the heart, that organ may beat without being able to transmit the impulse along the artery.

A pulse at once **unequal and irregular** is a much more serious indication than a merely intermittent pulse. It is observed especially in fatty degeneration of the muscular substance, and with imperfection of the valves on the left side of the organ, though it may be present in other cardiac diseases independent of the existence of those lesions.

In *hypertrophy of the left ventricle*, the pulse is full and strong and the impulse appears prolonged, because of the greater length of time taken up by the ventricle in the act of contraction. When *dilatation* coexists with *hypertrophy*, the impulse is still full and strong, more blood being transmitted through the vessel; but when *dilatation* is combined with *attenuation* of the ventricular walls the impulse is soft and weak by reason of the feebleness of the contractions.

The pulse at the radial artery should be about synchronous with the beat of the heart. If retarded it may be held to indicate the existence on the anterior aorta, or one of its primary divisions, of an aneurism with elastic walls, or more probably an imperfection of the aortic valves, which allows a regurgitation of the blood into the heart.

Venous pulse. A venous pulse seen in the lower end of the jugular veins is common in the domestic animals. In the ox it is quite compatible with health and is only to be judged by its amplitude and force. In other animals it often coexists with congestion of the lungs, which impedes the circulation through the right side of the heart and leads to engorgement of the venous system. In the absence of this condition it frequently indicates an imperfection of the auriculo-ventricular valves in the right heart and a reflux of blood from the contracting ventricle, which checks the descending current in the veins.

Percussion. In the horse a dull, dead sound is emitted, when percussion is made over the left side, for about four inches above the breast bone and in the space corresponding to the lower ends and the cartilages of prolongation of the fourth, fifth and sixth ribs. In the ox this dullness is less marked on the level of the sixth rib. The same results can be obtained on the right side by imparting heavier blows to the chest walls, so as to derive the sound from the deeper parts.

The area of dullness is *increased* in cases of *hypertrophy* or in *dilatation* of the heart, when the enlarged organ presses aside the lung tissue and exposes a greater amount of its substance to the chest walls. The same result takes place in hydropericardium.

The area of dullness is *diminished* in cases of ruptured air cells (as in "heaves") when the inflated and expanded lung tissue envelopes the heart more completely, and gives out its own clear resonance where the dull sound of the heart it usually obtained.

Application of the hand. Palpation. In conditions of health and in quietude the hand applied on the side of the chest, close behind the left elbow only just perceives the beat of the heart with each contraction. If the animal is excited, whether from fear, joy, or physical suffering the heart's impulse becomes more powerful and by this alone the state of its function may be very satisfactorily ascertained. The impulse is strong in all active fevers and extensive inflammations of important organs, but it is especially marked in diseases of the heart and lungs. Irregularity in the force of successive beats is seen in various heart diseases, and debilitated conditions are recognized in the same way.

Any want of harmony between the heart's action and the pulse may be observed by laying the right hand over the region of the heart, and applying the fingers of the left on the radial artery. In debility and especially if from a deficiency of blood, the violent or tumultuous action of the heart contrasts strangely with the weak, jerking compressible pulse. The same symptoms are noticed when the valves of the heart close the orifices imperfectly. In convalescence from lung diseases, and in certain diseases of the heart, a heart impulse may be felt by the right hand for which no corresponding pulsation is felt in the radial artery by the left.

When the heart is hypertrophied the impulse is stronger and is associated with a full, strong and rolling pulse. When it is atrophied the impulse on the chest and pulse beat are equally weak. When water exists in the pericardium the heart strikes the ribs with less force.

Sounds of the Heart. Synchronous with each beat of the heart two distinct sounds are heard, separated by a short interval inappreciable to most ears, and followed by a period of silence. These sounds are distinct alike in character and duration. The *first sound* is dull and prolonged; the *second* is short and quick. Some idea of these sounds may be formed by the pronunciation of the two syllables, *lub—tip*, but an acquaintance with the sounds themselves is essential to a correct conception of them. The period of time occupied by the first sound is double that taken up by the second, and in man and the smaller quadrupeds the subsequent period of silence is of equal duration with the second sound. Dividing the time belonging to one revolution of the heart into four equal periods, the first two are taken up by the first sound, the third by the second sound and the fourth by the interval of silence. In the horse the silence is more prolonged, and occupies the entire latter half of the period of a revolution. The relations stand thus:—the first sound extends over two-sixths of the time, the second sound over one-sixth, and the silence over three-sixths.

The *first sound*, synchronous with the beat of the heart against the ribs, corresponds also in point of time with the contraction of the ventricles, the closure and tension of the auriculo-ventricular valves, and the rush of the blood into the great arteries. The second sound corresponds to the reflux of blood in the arteries and the closure of the valves between them and the heart. The period of silence represents the period of rest, during which the heart is being filled from the veins.

In the horse, at rest, the first is the only sound that can be distinctly heard in many cases, but during the excitement of exercise or in febrile conditions the second is sufficiently apparent and any deviation from the natural character is easily noted.

These sounds are most distinct over the lower end of the fifth and sixth ribs on the left side, but they may be heard distinctly behind the middle of the shoulder on either side when the corre-

sponding limb is advanced. In birds they may be heard beneath the wings but above all and most clearly over the breast-bone.

In disease these sounds may be heard in unusual situations, they may be altered in force, duration or rhythm, or they may be associated with other sounds or superseded by them.

The sounds may be heard in new situations, in displacements of the heart from tumors or effusions in the chest, structural changes in the lungs, pleuræ, or pericardium, aneurism of the aorta, etc., etc.

The heart sounds are clearly heard over any part of the chest when the lung tissue intervening between that part of the surface and the heart is solid (hepatized). They are heard distinctly behind the median part of the right shoulder, when liquid effusion into the left pleural sac has displaced the heart to the right; and when the right cavities of the heart are extensively dilated, as exists so commonly in the advanced stages of "heaves."

The extent over which the sounds may be heard is *increased* when the lung surrounding the heart is solidified (hepatisation, splenisation, etc.), or when liquid effusion exists in the chest. A liquid but more especially a solid is a better conductor of sound than the spongy lung. Enlargement (hypertrophy) of the heart equally increases the area of sound. The *area* of sound is lessened by atrophy of the heart, and by emphysematous condition of the lungs by which the heart is more extensively covered and further separated from the walls of the chest.

The *force* or *intensity* of the heart sounds is increased in high fever, in acute inflammation, in increase of the muscular walls of the heart with enlargement of the internal cavities, in functional disturbance from fear or other exciting cause, and in palpitation. Often in a weak and bloodless patient, the heart sounds can be clearly heard at several yards distant from the animal. The *intensity* of the sounds is diminished in debility when not associated with palpitation, in atrophy of the muscular substance of the heart, in hypertrophy of the muscular tissue of the heart with diminution of its internal cavities, in heaves when the emphysematous lung more completely envelopes the heart, and in cases of extensive liquid effusion into the pericardium which prevents the apex of the heart from striking against the side of the chest.

The regular **rhythm**, normally manifested by the two sounds and the silence, may be modified in the unequal, irregular or intermittent contractions of the heart. Küssmaul's paradoxical pulse is one in which the pulse is more frequent but less full during inspiration than expiration. It is found in weak heart, during recovery from chest diseases, in chronic pericarditis, and when fibrous bands encircle the root of the aorta. Bigeminal and trigeminal when two or three beats follow each other rapidly, and are separated from the preceding and succeeding beats by longer intervals. This occurs in disease of the mitral valve, and in other weak states of the heart. Foetal heart rhythm in which the pause is shortened and the two sounds of the heart are almost simultaneous, is seen in the later stages of fevers, and in extreme dilatation. A curious aberration of rhythm is the *repetition* of either the first or second sound. If of the first sound (anapestic bruit) each beat will be accompanied by three sounds the first two of which resemble the first sound of health. If the second sound is repeated (dactylic bruit, bruit de galop) the first sound only will be prolonged and the last two sharp and quick. The repetition of the last sound is probably due to impaired nervous supply or hypertrophy of one ventricle which allows the completion of the contraction of the ventricle and the closure of the arterial (semi-lunar) valves sooner on one side than the other. If due to diminution of the arterial orifice which retarded the emptying of one of the ventricles, the first sound would probably be accompanied by a blowing murmur. If the auriculo-ventricular valves on one side were imperfect, allowing a reflux into the auricle and a more rapid emptying of the ventricle, a blowing murmur would equally accompany the first sound. In either of these two last mentioned cases the murmur would mask or hide the first of two doubled sounds.

The repetition of the 1st sound is often due to dilatation of one ventricle, which in consequence is longer in reaching the same sensation of plentitude, and in receiving the stimulus to contraction.

Morbid Sounds. Murmurs. The distinct and superadded sounds heard in disease are usually designated murmurs. They originate in the interior of the heart (endocardial) or externally to the heart (pericardial) The endocardial sounds mostly arise

from some abnormal condition of the valves or orifices and consist in a blowing or rushing noise which usually accompanies or displaces one of the heart sounds, though it may precede or succeed these. The following table, modified from that of Bartle and Roger, presents at a glance the relations of these different sounds and their significance.

Blowing or Hissing Murmurs.

Blowing murmur before the first sound.	----- {		Narrowing of the auriculo-ventricular orifice. Vegetations or coagula on the valves.
Blowing murmur with the first sound.	{		Strongest toward the base of the heart. Propagated along the great arteries.
	{		Narrowing of the aortic opening.
	{		Strongest toward the apex of the heart. Not propagated in the great arteries. No marked venous pulse.
	{		Narrowing of the pulmonary artery or insufficiency of the left auriculo- ventricular valves.
	{		Accompanied by venous pulse in the jugulars.
	{		Insufficiency of the tricuspid (right auriculo-ventri- cal) valve.
	{		No marked venous pulse in the jugulars: Congested lungs.
	{		Insufficiency of the mitral (left auri- culo-ventricular) valve
Blowing murmur with the second sound.	{		Double rushing sound heard over the great arteries, at each beat of the heart.
Blowing murmur after the second sound.	{		Insufficiency of the arterial (semi- lunar) valves.
	{		Double rushing sound in the arteries with each beat of the heart.
	{		Aneurism (Dilata- tion) of the great aorta.

From the table it will be seen that each orifice in the heart may become the seat of two perfectly distinct and independent murmurs: one due to constriction of the orifice in which case the sound is produced with the onward progress of the blood wave; and one due to dilatation of the orifice or insufficient closure of it by the valves, when the sound is due to a recoil or regurgitation of the blood. There is a further sound due to mere roughness of the valves in cases of disease when the sound will be with the normal rush of blood, though a second or regurgitant hiss is often heard from the valves being at the same time insufficient to close the orifice. Another blowing murmur is usually heard over the heart and coincident with its first sound in the bloodless state (anæmia). This is not necessarily connected with any diseased condition of the heart itself.

The nature of these murmurs differs in special instances. They may resemble the soft whisper of the words *who* or *awe*, of the double letter *ss*, or the single letter *r*, according as they are soft or hard and purring.

The pericardial murmur, caused by the rubbing of the dry roughened surface of the serous membrane covering the heart, on the correspondingly dry rough surface of the same membrane reflected on the investing sac, resembles that caused by passing the palm of the one hand softly over the other which lies on the ear.

It is distinguished from the friction sound of pleurisy by its coinciding with the movements of the heart and not with those of respiration. It is usually heard alike during the sounds of the heart and during the period of silence or in other words during the movement of contraction and dilatation in that organ.

General Symptoms of Heart Disease. In the acute inflammatory affections there are the signs of general constitutional disturbance attending similar affections in other organs. The decision as to the true nature of the disease must be arrived at from the special character of the pulse, heart sounds, etc., as already noticed.

In the chronic forms of the disease however a particular class of symptoms usually point towards the organ affected. In cattle, sheep and pigs raised only for slaughter, and as far as possible protected against active exertion, serious heart diseases may exist for a length of time without making themselves manifest by any prominent symptoms. Thus in cows, pins and other sharp pointed bodies, swallowed with the food, frequently make their way to the heart, and lodge for a length of time in its vicinity without material derangement, and when at last the animal dies a sudden death, they are found transfixing the walls of that organ. In the horse or other animal subjected to exertion, the symptoms are usually very patent.

When the heart is enlarged, the pulse strong and the circulation full and free, apoplexies or hemorrhages, especially on the brain or other soft organs where the resistance is least, are liable to occur. When on the other hand the circulation is weak from atrophy or fatty degeneration of the heart, or from insufficiency of the valves, there is a tendency to coldness of the extremities, and to passive congestions with their consequences :—serous effu-

sions, dropsies and difficult breathing. The imperfect supply of blood to the muscles of the extremities, sometimes brings about an unsteadiness of gait in the hind limbs when the animal is trotted for a short distance, and sometimes cramps supervene.

Continued coldness of the limbs, and a filling or thickening first of the hind limbs then of the fore and lastly of the chest and belly and of the skin beneath their dependent parts, are useful indications.

Shortness of breath and inability to proceed when trotted or galloped on soft, yielding ground, or when walked up hill, the animal being in fair condition, without fever or cough, but subject to cold extremities and a venous pulse in the jugulars, almost certainly indicates insufficiency of the auriculo-ventricular valves on the right side of the heart.

Vertigo, megrims, or giddiness may be caused by heart disease. The horse without having sustained any pressure on the veins of the neck by the collar, and having had no previous symptoms of brain disease, suddenly reels in harness and perhaps falls. There are the cold and engorged limbs or a tendency to their engorgement as in the former case. The attacks recur when the horse is put to the same exertion, and he proves utterly worthless. In such cases a careful examination of the pulse and heart sounds will complete the chain of evidence.

An almost constant feature of chronic heart disease is a condition of dullness, sluggishness, and in many cases, curiously enough, a tendency to lay on fat, so that although the patient is unfit to work he appears to enjoy excellent general health, to which a period is only put by sudden death.

Affections of the heart are primarily divisible into *functional* and *structural* disorders.

PALPITATIONS.

Convulsive contractions of the heart, functional or structural, diagnostic features of these. Significance of the functional disorder, genera most liable. Treatment, quiet, heart tonic, digitalis, correct other disorders.

These consist in a sudden violent and convulsive beating of the heart, not connected with any appreciable structural disease. They differ chiefly from the palpitations of organic disease of the heart in the absence of any apparent local change to account for their occurrence. The following table from Bellingham furnishes a number of criteria equally valuable in the lower animals as in man.

PALPITATION DEPENDENT ON ORGANIC DISEASE OF THE HEART.	PALPITATION INDEPENDENT OF ORGANIC DISEASE OF THE HEART.
1. Palpitation usually comes on slowly and gradually.	1. Palpitation usually sets in suddenly.
2. Palpitation constant, though more marked at one period than at another.	2. Palpitation not constant, having perfect intermissions.
3. Impulse of the heart usually stronger than natural, sometimes remarkably increased heaving and prolonged; at others irregular and unequal.	3. Impulse neither heaving nor prolonged; often abrupt and circumscribed knocking, and accompanied by a fluttering sensation (visible jerking or lifting) in the precordial region or epigastrium (flank and abdomen).
4. Palpitation often accompanied by the auscultatory signs of diseased valves.	4. Auscultatory signs of diseased valves absent; blowing sound often present in the large arteries and a continuous murmur in the veins.
5. Rhythm of the heart regular, irregular, or intermittent; its action not necessarily quickened.	5. Rhythm of heart usually regular, sometimes intermittent; its action generally more rapid than natural.
6. Mucous membranes often reddened and congested; dropsy of hind limbs common.	6. Mucous membranes generally pale; dropsy of hind limbs rare.
7. Palpitation increased by exercise, by stimulants and tonics, etc.; relieved by rest and frequently also by local and general bleeding and an antiphlogistic regimen.	7. Palpitation increased by close confinement; by local and general bleedings, etc.; relieved by moderate exercise and by stimulants and tonics, particularly the preparations of iron.

Palpitations in the lower animals, not dependent on any structural disease of the heart, are usually due to some violent mental emotion, such as fear or joy. The writer once possessed a fast and clever cob, having no sign of organic disease, and equal to the severest work on the road without showing signs of exhaustion, but which nevertheless was affected by palpitation, when threatened with a cane in his stall, or if he had been guilty of some wilful misdemeanor for which he dreaded punishment. In such cases the heart's action was accelerated and the beating could be heard loudly for a distance of several yards. They are especially common in dogs under strong mental emotion (joy, fear), and in dogs and pigs suffering from digestive disorder (worms), or chest diseases.

Percivall collects a series of cases from the *Veterinarian* under the head of *Spasm of the Diaphragm*, a diagnosis originating in the jerking movement of the flank, a symptom which, as is shown in the above table, is common to man as well. Leblanc equally collects cases from the French veterinary journals and acknowledges their true character. One of these observed by Coulbeaux is thus described: "The respiratory movements are interrupted by a violent lifting of the flanks, confined to the upper part, and so intense as to be appreciated by the hand as well as the eye. The lifting of the flank which is limited to a few square inches of surface precisely in the hollow of this region, is perfectly isochronous with the beats of the heart, which cannot be appreciated but by the ear."

A case reported by Levrat at greater length may also be given. The subject a mare, fifteen years old, and kept for hire, was attacked without any known cause. "There was a jerking of the whole body produced by the extraordinary force of the beats of the heart which struck violently against the back towards the upper ends of the first asternal ribs on the left side. The shock is very distinct and does not appear to be due to any tremor interposed between the heart and the ribs; its force is such that it causes a movement of the whole body, which can be seen at a great distance. The beats of the heart very regular, numbering fifty per minute, were heterochronous with the pulsations of the submaxillary artery, which were of the same number." (The pulsation was evidently retarded). "The

pulse was soft and regular. The breathing was peculiar. On approaching the ear to the nostrils the animal is heard to make three successive inspirations which coincide with the beats of the heart; each of these inspirations is followed by an expiration so short and feeble that the expired air can not be felt; the fourth is followed by full expiration sustained during three beats of the heart." This form of respiration was continued without intermission for an entire day.

In three cases observed at the Lyons Veterinary School the disease continued for eight days. In all these and twenty more observed by Leblanc, the patients invariably recovered. The steady persistence of the disease for several days and the subsequent complete recovery under the unaided action of digitalis would seem to warrant the conclusion that such cases were really accompaniments of structural diseases of the heart and not mere functional disorders. Even inflammation of the lining membrane of the heart often exists without any obvious fever or other manifest symptom of illness, and in the dissecting rooms of medical schools nothing is more common than to find traces of pre-existing heart disease in patients whose whole life had been passed without the suspicion of such a malady.

A number of such cases observed in England and on the European Continent are adduced to prove spasm of the diaphragm or of the abdominal muscles, (Delafond), and great importance is attached to the fact that the conclusive movements of the abdomen and loins are heterochronous with the beats of the heart. This lack of exact coincidence, however, does not seem to amount to more than a perceptible delay after the heart beat, just as the maxillary pulsation is delayed in case of aneurism of the aorta. This has been my own experience with such cases. The flank movements have been equal in number to the heart beats or have corresponded to certain beats in the heart cycle, and have been perceptibly retarded in accordance with the necessity for time for the transmission of the blood wave along the posterior aorta, and the development of the reflex action which set the phrenic and abdominal muscles in motion. We must of course accept the convulsive action of the phrenic and abdominal muscles, only it would seem that each such movement has its starting point in the contraction of the heart. In cases that show no relation in number

nor succession to the heart beats, we can freely acknowledge a neurosis starting at a point different from the heart, but in all examples which are manifestly connected with heart movements, and readily curable by the heart-tonic, digitalis, the recognition of the cardiac derangement as an initial factor is sound alike in pathology and practice.

Nervous and purely functional palpitation are probably confined to cases in which they are manifested at intervals, appearing under the influence of some transient excitement and continuing only for a few minutes at a time.

In the anæmic or bloodless condition the palpitation of weakness is often observed under the slightest exciting cause. It is then associated with a pallid state of the visible mucous membranes, a weak, trickling pulse and a blowing murmur in the larger veins.

Palpitations are much more frequent in pigs and dogs than in horses.

Treatment. Quietness and avoidance of all excitement are first to be secured, then the action of the heart is to be calmed and regulated by the use of digitalis. To the horse 15 to 30 grains of the powdered leaves may be given thrice daily, and to the dog from two to four grains at equal intervals. When the disease is associated with anæmia this agent may still be used in combination with the various tonic remedies recommended, but in the smallest doses only, as the heart is usually morbidly sensitive to external influences. Chloral hydrate has been used with success. Bromides, valerian, and belladonna may be tried in obstinate cases. Any other deviation from a healthy condition must be noted and corrected, especially any disordered condition of the stomach or bowels.

ANGINA PECTORIS. BREAST PANG.

W. Williams describes a case of illness in the horse to which he gives this name. When standing idle he had twitchings of the pectoral muscles, and when exercised these and adjacent muscles became violently convulsed, the left fore limb being alternately fixed by spasm, and paralyzed so that it was useless and the animal fell if compelled to move. There was "venous pulse, great irregularity of the heart's action, a loud cooing or blowing sound and strong impulse indicative of hypertrophy and a want of correspondence between the cardiac energy and feeble pulse." It was unfortunate that the murmur was not associated by the observer with a particular heart sound, and with the right or left side of the heart, and that hypertrophy was not diagnosed by percussion, since the case can be of little value as it stands.

Breast pang is usually associated with some disease of the heart; obstruction (usually calcification) of the coronary arteries, insufficiency of the aortic valves, calcareous degeneration of the aorta, aortic aneurism, or fatty degeneration of the heart. Loomis, basing his view on dissections made by himself and others, resolves all of these into concurrent ischæmia of the heart, the circulation in the coronary arteries being seriously interfered with. "That the sudden withdrawal of the supply of blood to a part may occasion neuralgia is shown by the intense pain in the limb, which directly follows embolism of the femoral artery. Moreover, general anæmia, as is well known, favors the recurrence of neuralgia in various situations." Inability of the heart to propel the blood is to be explained in the same way.

The difficulty of endorsing Williams' diagnosis lies in the fact that the disease, so far as it is a distinct disease, is functional and manifested by pain, the nature of which can only be inferred in the case of the lower animals (not by spasms of the pectoral muscles), and that hypertrophy of the heart is not likely to be present in case of insufficiency of blood supply to its walls.

In true angina pectoris of the horse, treatment is useless. Absolute rest is a prime requisite, and anodynes, stimulants, heart tonics, and nerve tonics are indicated. But the horse at rest, with no prospect of final recovery, is simply a source of expense.

Cases have been noted in the *horse* in connection with obstruction of the coronary artery by sclerostoma equinum (larva) (Cadiot), with a microbial thrombus of the coronary artery following tenotomy (Johne), and with rupture of the right coronary artery (Rigollat), and in the *ox* with aneurism of the left coronary artery (Piot). Other cases in *horses* have been recorded by Cagny and Butti.

Symptoms. Coming as it usually does from imperfect blood supply to the walls of the heart, the symptoms are essentially those of heart failure and profound prostration under active exertion or excitement. The animal will suddenly stop work, whinny plaintively, standing stock still with a drawn, anxious face, and drooping head. In some cases it is impossible to move him; in others he may be moved a few steps at a time, with rests of about five minutes each, which bring relief. Auscultation detects irregular action of the heart, with the sounds low and indistinct. The pulse is weak, thready and irregular, so that it cannot be certainly counted. Respiration is deep. In some cases the patient suddenly drops to the ground, and may perish or revive. The attacks are paroxysmal, when not fatal they recur at intervals of a few days up to a year, but tend to greater frequency and finally to sudden death in a paroxysm.

Treatment. Give choral by stomach or rectum; amyle nitrite by inhalation or morphine sub-cutem.

FUNCTIONAL IRREGULARITY IN THE RHYTHM OF THE HEART.

Associated or not with palpitation, irregularity in the force or frequency of the heart-beats is sometimes met with at intervals or independently of any further indication of structural disease. Particularly in the greyhound and certain other breeds of dogs, the temporary occurrence of intermitting action of the heart is a frequent though a very transient condition. It may be excited by some emotion or excitement such as the attentions of the owner, or the straining anxiety in the immediate anticipation of the chase. Here again digitalis is pronounced the great panacea though it need not be resorted to unless the habit interferes with the usefulness of the animal. If in any degree dependent on weakness, that must be counteracted by a systemic tonic treatment.

CONGENITAL MALFORMATIONS AND DISPLACEMENTS OF THE HEART.

Ectopia Cordis. Cyanosis, previous foramen ovale. Symptoms: blue mucosæ, coldness, staring coat, unthriftiness unequal to exertion, palpitations, murmur before the first heart sound. Obstructed circulation in the lungs as a cause of cyanosis, cyanosis as a cause of lung disease.

These have been much less frequently observed in the lower animals than in man. The anomalies observed in mammals include the following: the displacement of the heart to the right side of the chest; displacement entirely out of the chest (ectopia cordis); permanent communication between the right and left auricles (cyanosis); entire absence of heart; two hearts; one common ventricle communicating with two auricles, as in reptiles; three ventricles; only one auricle; absence of one or several valves; absence of the pericardium; variations in the mode of connection of the heart and large vessels, etc.

Displacements of the heart have been especially studied by Hering, on calves. The breast bone remained as originally developed in two lateral halves, and the heart remained outside, connected with the interior of the chest only by its large vessels. The heart thus exposed and covered only by its investing membrane (pericardium) afforded an excellent opportunity to study its action, of which Hering freely availed himself. Animals affected in this way survived their birth but a very short time. An approach to this condition was, sixty years ago, made familiar to the medical world in the person of M. Gouz, a German mechanic, the movements of whose heart could be easily watched through a fissure in the breast bone.

Permanent communication between the two auricles. Previous foramen ovale. Cyanosis. Previous to birth there is an opening between the right and left auricle, allowing the blood to flow from the former into the latter in place of, as in after life, descending into the right ventricle and thence circulating through the lungs. At birth this is contracted, and in a few days is completely closed in accordance with the new life, which demands that all blood must circulate through the lungs in order to secure its aëration. Sometimes this closure fails to

occur, and venous blood from the right side of the heart continues to mix with arterial in the left, deteriorating it in quality and unfitting it for nutrition, secretion, calorification and other essential processes. The semi-venous blood circulating in the arteries gives a bluish hue to the visible mucous membranes, hence the name of *the blue disease*. This blood is unfit for sustaining the vital changes essential to the production of animal heat, so that the animal suffers from coldness of the surface and extremities, staring coat and general unthrifty appearance. Such subjects grow badly, and refuse to lay on flesh, but are said to arrive at maturity in some instances and to have their imperfection recognized only because of the short breathing and irregular heart's action when subjected to exertion. A heart murmur preceding the first sound of health is usually present, as in anæmia.

Drs. Abernethy and Wardrop draw attention to the frequency of pervious *foramen ovale* in the human subject in connection with pulmonary consumption, and opine that it is reopened as a consequence of this disease. The coincidence has not been observed in the lower animals, though, if it were found to exist, the question would arise whether the deterioration of the blood and general health in open *foramen ovale* did not also favor the deposit of tubercle in the lungs. When from deficient ventilation the atmosphere and blood become impregnated with carbon dioxide, the production of tubercle in man or in animals is correspondingly frequent.

The subjects of previous *foramen ovale* die young or prove worthless when they arrive at maturity. Nothing can be done to ameliorate the condition.

HYPERTROPHY OF THE HEART.

Simple, eccentric, concentric. Ventricles chiefly affected. Causes: increased functional activity, from obstruction to the circulation, or continued extra exertion. Right ventricular hypertrophy-obstruction in the pulmonary circulation; left ventricular hypertrophy-obstruction in the systemic. Auricular hypertrophy-insufficiency of the auriculo-ventricular valves. Pericarditis as a cause. Abnormal weights. Symptoms: beats more forcible and prolonged, 1st sound low, prolonged, 2nd sound clear, often doubled, increased dullness on percussion, diagnostic signs of hypertrophy, dilatation and a combination of the two. Simple hypertrophy rarely dangerous, with dilatation grave, threatens congestions and apoplexies. Treatment: rest, laxatives, sedatives, in irregular heart action digitalis, arsenic.

An enlargement of the heart from increase of its muscular substance is by no means uncommon in the horse. It may exist without any change in the capacity of the cavities of the heart (simple hypertrophy) or it may be associated with dilatation of one or more of these cavities (hypertrophy with dilatation;—excentric hypertrophy). A third variety has been described in which the capacity of the cavities is decreased, but Cruveilhier and Budd have satisfactorily shown the non-existence of this condition except as a congenital deformity.

It is in the ventricles that the increase is chiefly observed, the reason of which is to be found in the causes of the malady. These usually consist in some obstruction to the circulation such as chronic congestions in the lungs or elsewhere, rupture of air cells in the lungs, tuberculous and other abnormal deposits in the chest and elsewhere, tumors which by their position interfere with the circulation through the larger vessels, and the like. Where by some such cause the blood is impeded in its outward course, one or both ventricles are called upon to contract more vigorously to force a sufficient amount of blood onward and, in accordance with the inherent adaptability of the animal economy, there takes place an increase of the muscular walls of the ventricle, proportionate to the required energy of the contractions. The condition then is essentially due to a more active nutrition and growth of the muscular substance and finds its exact parallels in the well-developed legs of the ballet dancer or the brawny arm of the blacksmith. All alike occur in accordance with a general

law that whenever there is habitually demanded of any organ an unusual activity of function, which stimulates without exhausting its power, nature adds to the active element of such organ till the required labor can be accomplished without the overwork of any particular part.

Keeping this in view we can easily explain the increase of one part of the heart without immediate implication of another. The ventricles are more commonly enlarged than the auricles because upon them devolves the work of overcoming the obstruction, whether this exists in the lungs or the system at large. The auricles fulfill little more than a passive function in receiving the blood from the veins during the contraction of the ventricles and allowing it to pass down into these ventricles when their relaxation takes place. The closure of the auriculo-ventricular valves during the ventricular contraction protects the auricles from the internal tension to which the lower part of the heart is subjected, and thus all tendency to increase is obviated.

The hypertrophied part corresponds to the locality of the obstruction. If it exists in the lungs (heaves, consumption, hepatisation, chronic bronchitis), pulmonary artery, its valves at its origin from the heart, or if it consists in contraction of that orifice, the enlargement takes place primarily in the right ventricle, the right auricle remaining unchanged so long as the auriculo-ventricular valves act perfectly. The ventricle, however, tends to dilate as well as enlarge in thickness of walls, and as soon as this dilatation has proceeded so far as to widen the orifice between the auricle and ventricle and render its valves insufficient, the auricle also begins to dilate and its walls often increase in thickness. But the vicious chain does not end here. Should the animal survive and the original obstruction persist, the veins throughout the system become habitually congested because of the reflux of blood from the right auricle and ventricle, dropsies appear in different parts, the congestion of the veins is continued through the capillary blood vessels to the arteries, the difficulty of propelling the blood comes to be experienced by the left ventricle and a corresponding series of morbid changes taking place on that side, as has already ensued on the right, the vicious circle is soon completed, the entire organ becomes diseased, each constituent part of the organ operating injuriously on that

which preceded it in the track of the circulation, and every new change forming but a stepping stone to a more dangerous modification.

On the other hand, the obstruction may exist in the general circulation, on the course of the aorta, or its branches, in its valves at its origin from the heart, or in the narrowing of its orifice. Then the increase takes place first in the left ventricle, is propagated to the left auricle, leads to congestion of the veins, capillaries and arteries of the lungs, and lastly to disease of the right side of the heart. Here there is a different starting point, but the progress of the disease-changes, in a direction opposed to the course of the circulation, is the same.

The disease may, however, begin with the auricles, owing to disease of the auriculo-ventricular valves impeding the flow of blood into the ventricle, or to simple narrowing of the auriculo-ventricular opening. The auricle is then primarily enlarged, the corresponding veins congested, this is propagated to the capillaries and arteries, and lastly the ventricle on the opposite side of the heart is involved. This is chiefly seen with fibrinous deposits on the valves or in the case of polypus hanging into the auriculo-ventricle opening. Aneurisms, embolisms, neoplasms, atheroma and calcic degeneration of the arterial walls may be effective factors.

In addition to these causes, Bouilaud and Leblanc attach a high importance to chronic inflammations of the serous membranes, which, by reason of the contiguity of the latter to the muscular structure, bring about a more abundant circulation in this and an increased nutrition. Another cause is unintermitting hard work which necessitates excessive exertion of the heart, to supply blood more freely to the muscular system and the lungs. Many hunters suffer from this affection, it is believed because of their extraordinary exertions. The stallion Helenus had a heart of 14 lbs.

Weight of the heart. The heart in the horse which rarely weighs over 9 lbs. is increased from 10 lbs. to 14 lbs. in this disease and in one case in a cart horse, recorded by Stephenson in the *Veterinarian* for 1861, it is said to have reached 32 lbs. Stephenson probably weighed the heart while filled with blood. A diseased heart weighed in this way by Thompson amounted to

34 lbs., one by Gerlach, 19 lbs., an ox's heart by Herran, 36 lbs. In Stephenson's case there was further an extraordinary dilatation of the anterior vena cava. Haycock (Veterinarian, 1850), records a case in which though the heart only weighed 10 lbs. 8 oz., yet the walls of the ventricles were double the normal thickness, those of the left being $2\frac{1}{4}$ inches, while those of the right were 1 inch. An estimate from the thickness of the walls, it must be borne in mind, is not so satisfactory as the absolute weight taken after the removal of the large vessels, the superfluous fat and the contained blood.

Horse. *Causes of Hypertrophy.* The following conditions in different cases proved active factors in causing hypertrophy: chronic endocarditis with insufficiency of the auriculo-ventricular valves (Schaffer, Haycock, Hill); stenosis of the arterial orifices (Trasbot, Lustig, Osterman, Cadeac); exostosis on tenth and eleventh vertebræ pressing on the posterior aorta (Pirl); extensive melanotic deposits in the spleen, liver, kidneys and mesenteric glands; (Halloway); aneurism of the femoral artery and insufficiency of the aortic valves (Gueripell); sclerosis of the walls of the posterior aorta (Lascaux); stenosis of the right renal artery and atrophy of the kidney (Cadeac); thrombosis of the iliac arteries (Cadeac); emphysema, chronic (fibrous) pneumonia, tuberculosis (Cadeac); pleurisy, chronic hydrothorax (Percivall, Cadeac); hypertrophy of the liver (Henderson); exophthalmic goitre (Cadiot); gestation (Cadeac); myocarditis, pericarditis.

Cattle. *Causes of hypertrophy.* Cattle take less compulsory exertion and suffer less from hypertrophy. Tuberculosis and emphysema are the most common causes, but it has coincided with a perforating opening in the septum ventriculorum (Eisenblätter), and myxoma of the septum auriculorum (Koch).

Dog. *Causes of hypertrophy.* Like other heart diseases this is common to dogs. It occurs from habitual violent exertion; hepatitis; tuberculosis of the lung; valvular insufficiency; aneurisms of the aorta from spiroptera sanguinolenta (Cadeac); aneurism of the pulmonary arteries from filaria immitis (Law, Cadeac); myocarditis (Cadeac); and, it is alleged, frequent and prolonged generic excitement (Fricker).

Birds. *Causes of hypertrophy.* The condition is said to be

common in pigeons, and has been attributed to natural timidity, excessive emotion and sudden excited movements (Cadeac).

Symptoms. In simple hypertrophy the heart beats are more forcible and prolonged so that the period of silence or rest is shortened. This is due to the greater length of time taken up in the contraction of the ventricles. For the same reason the pulse which may or may not be accelerated, irregular, or intermittent, is full and rolling or as it were prolonged. The first sound of the heart is prolonged and low or muffled, sometimes almost inaudible, while the second is unnaturally loud. Sometimes when one ventricle only is enlarged that may complete its contraction later than the other and the second sound is repeated as in the syllables *lub—tip tip*. A duplication of the first sound only is less common. If the sounds are heard over a greater extent of the chest's surface than is natural, the lungs being healthy, it is probably due to hypertrophy of the heart. If very clear on the right side they indicate increase of the right ventricle. The heart's impulse is usually strong and may be felt on both sides, and it may be over the whole chest.

Percussion usually shows a more extended dullness in the region of the heart, but the blows must be pretty forcible to bring out the deeper resonance, otherwise it will come only from the thin layer of lung. These results are of the greatest value in the dog.

The pulse is usually regular and if excited to irregularity and intermission, quickly returns to its natural state when the patient is left at rest.

As hypertrophy is usually associated with dilatation of the heart, the following table will prove valuable by presenting side by side the signs indicating hypertrophy with and without dilatation, and simple dilatation.

TABLE CONTRASTING THE MAIN SYMPTOMS OF HYPERTROPHY
AND DILATATION.

A. General Physical Signs.

SIMPLE HYPERTROPHY.	HYPERTROPHY WITH DILATATION.	SIMPLE DILATATION.
Heart's impulse slow and heaving as if pressing steadily against an obstacle — in rhythm regular, in force unequal.	Force increased, sharper, more knocking, may impart a shake to the body. May be felt on the right side.	Impulse conveys a feeble undulatory sensation; force of successive beats unequal; rhythm irregular.
First sound is dull, muffled, prolonged and weakened almost to extinction. Second sound full and clanging; period of silence shortened.	Sounds gain greatly in loudness and extent of transmission, especially if the valves are not thickened.	First sound short, abrupt and clear. Second sound not specially affected.
Murmur with the first sound present at one time and absent at another.	Murmur with the first sound may be present, from altered direction of the orifice of the aorta, or from insufficiency of auriculo ventricular valves. With second sound from insufficiency of arterial valves.	Murmur with the first sound from inefficiency of the auriculo-ventricular valves; or with the second with insufficiency of the arterial valves.

B. General Functional Symptoms.

SIMPLE HYPERTROPHY.	HYPERTROPHY WITH DILATATION.	SIMPLE DILATATION.
Strength unimpaired. Power of continued exertion (especially uphill) limited by shortness of breath.	Strength tends to become impaired.	Strength fails.
Visible mucous membranes, healthy or of a bright red.	Purpleness and lividity of the mucous membranes proportionate to the valvular or pulmonary obstructions.	Lividity of the mucous membranes. Dropsical effusions of the limbs and other dependent parts, which pit on pressure.
Difficulty of breathing occasional.	Difficulty of breathing occurs in paroxysms.	Difficulty of breathing great and constant, with occasional aggravations

SIMPLE HYPERTROPHY.	HYPERTROPHY With DILATATION.	SIMPLE DILATATION,
Pulse full, strong, firm, tense, resisting and prolonged without jerk or thrill.	Fullness of pulse continues but strength and power of resistance lost.	Pulse small and feeble, much later than the heart beat. Regular or feeble, fluttering and irregular. Venous pulse in the jugulars.
Rarely and never rapidly the direct cause of death.	Indirectly and more or less rapidly fatal.	Palpitation frequent. Faintness occurs from time to time, and may lapse into fainting and sudden death.

Pure hypertrophy rarely implies imminent danger unless dependent on some pre-existing structural disease which impedes the freedom of the circulation. If excessive, however, or if associated with dilatation, the animal is short-winded and unfit for all but the slowest work. It predisposes to congestion or apoplexy of the lungs when its seat is the right ventricle, and to congestions and hemorrhage in other parts of the system, brain, kidney, lungs, liver, bowels, if in the left.

Asthma (dogs), heaves (horses), emphysema and tuberculosis in cattle are occasional complications attended by grave symptoms.

The *dog* is liable to show the symptoms of valvular insufficiency: dyspnoea, giddiness, impaired eyesight, palpitations; also forcible heart impulse, loudness of the first heart sound, and low muffled second sound, blowing sound opposite the middle or base of the heart on the left side and during deep inspiration, and cardiac murmur with either first or second sound. There is an irregularity of heart rhythm and pulse. Finally, percussion shows an increased area of flatness, not only observed over the sternum and breast, but also on the lateral thoracic walls, and often extending as far as the ensiform cartilage..

In *pigeons* the violent palpitations shake the body, and there may be hemorrhages:

Treatment. In advanced cases and such as are dependent on irremovable structural changes in the lungs or elsewhere, no treatment is of any avail. In recent and uncomplicated cases in the horse and cow and in some more advanced conditions in other animals, not used for work, a palliative treatment may be

profitably adopted. This consists in a nitrogenous restricted and gently laxative diet, perfect rest in oxen and other fattening animals, or in the horse moderate and carefully regulated work, and as a medicament the use of digitalis or aconite. No known remedy has any power to directly check the growth of the heart and the utmost that can be expected of these agents is to lessen the activity of the heart's action and retard its growth. Digitalis may be given as recommended for palpitation, or aconite in the form of tincture, 20 drops for horses and cattle and 1 to 2 drops for dogs, repeated four times daily. Strophanthus may replace digitalis. When depletion seems advisable, purgatives or diuretics should be given as appears most applicable to the particular case. Iodide of potassium has been strongly recommended.

When extreme dilatation exists with the hypertrophy, sedatives should be given cautiously and their effects carefully watched, as the heart is often dangerously susceptible to depressing influences, When the disease has advanced so far as to cause abundant dropsical effusions, it is futile to resort to treatment, as amelioration can rarely be looked for, not even to the extent of allowing an animal to be fattened.

The value of arsenic in most cases of broken wind (*heaves*) has suggested the inquiry whether it does not operate directly on the heart, the known power of arsenic to retard and arrest tissue change, the decrease of effete matter thrown into the blood in any given time, and the lessened necessity for an active circulation to supply any great waste of structure being noted. It may benefit such cases in this way but does so probably to a far greater extent by an influence on the nervous function, analogous to its action in neuralgia and other purely nervous disorders. Dilatation of the heart which usually exists in *heaves* is commonly benefited by tonics which, like arsenic, are destitute of stimulating properties.

ATROPHY.

Simple, eccentric, concentric. Usually eccentric. Causes: effusion in pericardium, obstruction of coronary arteries, by false membranes, etc., general inanition. Symptoms: beats weak, sounds loud, clear, decreased area of dullness on percussion, pulse slow, weak, under excitement unequal, irregular, intermittent with palpitation, dropsy of limbs, etc., murmur with 1st sound. Treatment only in early stages by removal of the cause.

The loss of substance in the muscular walls of the heart is either **simple** when there is no change in the capacity of its different cavities;—**eccentric** when the chambers of the heart are enlarged; or **concentric** when these chambers are reduced in size. Like hypertrophy it may affect the walls of one chamber to the exclusion of the others.

Atrophy is much less frequent in the lower animals than hypertrophy and in nearly all cases on record it was associated with dilatation.

The *causes* are not always very evident. Effusion into the pericardium is one of the most frequent, the compression of the heart impairing its nutrition and decreasing its size. Especially is it hurtful when several layers of false membrane deposited on the surface of the heart become organized, preventing its sufficient dilatation and compressing its nutrient blood-vessels. A case of this kind in a dog is noted by Leblanc; the right auriculo-ventricular opening was surrounded by thick organized layers of false membranes which, by their contraction, had largely diminished the opening and even pressed on the coronary artery, cutting off to a great extent the supply of blood to the walls of the ventricle. Another cause in dogs, etc., is a prolonged insufficient nourishment of the entire body.

Symptoms. In pure atrophy these are the opposite of those seen in hypertrophy. The beats of the heart are weak or inappreciable to the hand placed on the side of the chest behind the left elbow. The sounds of the heart are loud and clear, their intensity being proportionate to the thinning of the walls and the dilatation of the chambers. Percussion so far as it can be made effectual, which is chiefly in dogs, shows a diminished area of

dullness. The pulse is slow, weak, or indistinct, compressible, becoming accelerated, unequal, irregular, and intermittent when the patient is excited. Palpitation is frequent, breathing is difficult or easily embarrassed and there is a tendency to dropsy of the limbs and dependent parts. These symptoms are usually associated with considerable prostration and depression.

These are often complicated by symptoms of valvular disease or dilatation,

Atrophy progresses slowly and rarely causes death in the earlier stages. In its advanced stages, when dropsy has supervened, little can be done even in its mitigation. In the earliest stages only, can good be done by employing measures calculated to remove its causes and thus put a stop to its progress.

DILATATION OF THE HEART.

Result of obstruction to circulation. In right ventricle usually. In auricle from narrow auriculo-ventricular opening. Pure dilatation from sudden extreme blood pressure as in inflammations of the lungs. In fat cattle from fatty obstructions around the heart and great vessels. Weakness of cardiac muscles in fatty degeneration, fevers, debility, etc. Symptoms : dyspnoea under slight exertion, unsteady walk, cold, dropsical limbs, venous pulse, pulse small, weak, irregular, intermittent, with palpitations. Treatment : in early stages arrest the causes, arsenic, digitalis, fatten for butcher.

Dilatation of the right cavities of the heart is one of the most common heart diseases of the horse. It is an almost constant condition in the emphysema of advanced heaves, and is a frequent concomitant of hypertrophy and an occasional one of atrophy of the heart. Its usual direct cause is some obstacle to the free escape of blood from the cavity affected. Thus, in heaves, the difficulty of the circulation through the lungs causes accumulation in the pulmonary artery and right ventricle of the heart, the walls of which are distended because of the unwonted internal pressure. When the dilatation of this ventricle reaches a certain stage the auriculo-ventricular opening is equally widened, the valves become insufficient to close it and the right auricle and venæ cavæ participate in turn in the internal pressure and dilatation. The right ventricle is more often affected than the left, because of the greater frequency of obstruction in the pulmonary than the systemic circulation, and because of the thinness of its walls which more readily give way under internal pressure. Dilatation may result from disease of the great arteries, from diminution of their calibre by the pressure of tumors, or by narrowing of their openings at the heart, whether as the result of diseased valves or other morbid condition. As affecting the auricles primarily its usual cause is narrowing of the auriculo-ventricular opening from some abnormal deposit. The extreme thinness of the walls of the auricles allows these to give way under internal pressure, even much more readily than the right ventricle.

The causes it will be seen are similar to those inducing hypertrophy, and hence the frequent co-existence of the two. Pure dilatation occurs especially when internal pressure takes place suddenly and to excess, and while the nutritive functions are to a great extent in abeyance. Such conditions are found in acute inflammations of the respiratory organs, or of the inner or outer membranes of the heart, and the rapid deposit in the lungs of tubercles or other abnormal material.

Dilatation of the right side of the heart is a common complaint in overfed *cattle*, and is apparently due to the diminished power of resistance in the fatty walls of the heart, and to the obstruction offered to the circulation by the extraordinary accumulation of fat around the base of the heart and the commencement of the large blood vessels. Though a diseased condition, this rarely shortens life or interferes with the use to which cattle are put. Pulmonary tuberculosis is a common cause, the blood being forced back on the right ventricle.

Chronic bronchitis or pneumonia. The heart walls are similarly weakened and yield more readily to the internal blood pressure in endocarditis, myocarditis, pericarditis, high fever, infectious diseases, poisonings, anæmia, and debilitating diseases generally. Debility, degeneration and incapacity to resist the blood pressure are essential prerequisites to dilatation. Old age is a common factor in dogs.

The *symptoms* which have been already enumerated in the table given under the head of *hypertrophy* are mainly these: Loss of appetite, spirit and endurance; faintness and difficulty of breathing on the slightest exertion; habitual coldness with a tendency to dropsy of the extremities; ascites; hydrothorax; loss of control over the extremities when walked or trotted far; venous pulsation in the jugulars; heart's impulse weak and undulatory or tremulous, or under exertion tumultuous or palpitating; murmur often present with the first sound; the first or more commonly the second sound may be doubled; pulse small, weak, irregular, and often intermittent, and frequently livid spots in the nasal mucous membrane. Paroxysms of unsteady gait from irregular circulation in the brain are frequent, and Dyer asserts that he has repeatedly seen blindness as a result of this condition.

In *treatment* the main purpose should be to put a stop to the

cause of the disease before it has been developed to a dangerous extent. When the malady is manifested by the symptoms above enumerated the subject is rendered permanently unfit for service and will probably die suddenly under some slight exertion. Fattening animals in a condition of quietude will often lay on flesh for an indefinite length of time, notwithstanding that the heart is considerably dilated. (See note on digitalis, strophanthus and arsenious acid in dilated heart, under the head of *hyperthrophy*). To relieve the asthmatic attacks attending on an overtaxed heart Zuill strongly recommends the combination of iodide of potassium, digitalis, nux vomica and coca. But heart tonics are often much more effective after the bowels and portal system have been unloaded by a laxative.

SERO-FIBRINOUS PERICARDITIS.

Definition. Frequency in different genera. Causes, rheumatic, traumatism, extension from pleurisy. Unwholesome buildings and localities, debility. Symptoms, chill, reaction, pleuritic symptoms, hyperthermia tenderness behind left elbow only, friction sound synchronous with heart beat, later it is lost and heart sounds are muffled, increased area of dullness on percussion, oppressed breathing, venous pulse, patient statant, little fever in chronic cases. Traumatism from the stomach: digestive disorder grunting, dropsy under the sternum with little fever at first. Lesions as in pleurisy, obliteration of pericardial sac. In traumatism from stomach the foreign body is found in the cardiac end of a band of lymph extending to the reticulum. Treatment, in chill, after reaction, medicinal measures as in pleurisy, local applications to the region of the heart. Paracentesis, insertion of needle, antiseptic precautions. Chronic pericarditis in oxen.

Definition. Inflammation of the strong fibro-serous sac in which the heart is contained and which is reflected on the muscular substance of that organ so as to form its external covering.

This is the most common inflammatory disease of the heart and has been met with more frequently in horses and cattle than in the smaller quadrupeds.

Causes. It is frequently a secondary disease and coincides with or follows other infectious diseases such as *influenza*, *pleuropneumonia* and above all *rheumatism*. In cattle and goats, wounds from sharp pointed bodies, (needles, pins, nails, etc.), which have been swallowed with the food and have passed through the walls of the second stomach, the diaphragm and pleura to the heart, constitute a frequent cause. The pericardium has been punctured by a fractured rib and has been implicated in inflammation attendant on an abscess or other lesion in the walls of the chest. Kicks, bruises, concussions, injure the heart, laying it open to microbial attack. Besides these the general influences which cause primary and uncomplicated attacks of the disease are the same as those producing pleurisy, peritonitis, rheumatism and inflammation of serous membranes generally. These are sudden changes from heat to cold, cold winds, cold draughts, drenching, chilling rains in animals already overheated and exhausted, or prolonged exposure in severe weather, in low states of the system. Leblanc justly remarks that "with the morbid influences which appear specific, there often coincide intemperate seasons, badly

arranged buildings, a want of sufficient attention to the conditions of health, and in the case of herbivora, wet, cold, and badly exposed pastures." In other words whatever deteriorates the health and vitality predisposes, paving the way for microbial invasion.

Given thoracic epizootics attack the heart often, others, or the same a few months later, respect it (Dumas). *Cattle* suffer through blows with the horns of themselves or others, through compression in a partly closed gate, through rheumatism, lung plague, contagious pneumonia, tubercle, etc. *Oxen* suffer through chills after heating, exhausting work.

In *sheep* acute infectious pericarditis, may be but a form of hæmorrhagic septicaemia, malignant oedema, or emphysematous anthrax. Again it may be parasitic (*Echinococcus*, *Cysticercus Tenuicollis*).

In *pigs* it is often but a manifestation of a prevailing infection (swine plague, rouget, tuberculosis, rheumatism,) or parasitism (*cysticercus cellulosa*, *trichina*, *echinococcus*).

Dogs have primary pericarditis from blows, kicks, bites, goring, shot, etc., and from chills in an exhausted system, but far more commonly it is microbial (tuberculosis, distemper, pyæmia). Tumors too are a common cause.

Symptoms: These are less characteristic than in man owing to the smaller portion of the heart exposed, but they are usually marked enough to permit a recognition of the disease. *Acute form.* The affection is ushered in by a chill, general fever, hyperthermia, (103° to 104°), staring coat, hot, dry mouth, dilated nostrils, excited, difficult breathing, double lifting of the flank with each expiration, the existence of a prominent ridge from the lower end of the last ribs along the flank to the outer angle of the hip bone, as in pleurisy, pinched, anxious expression of countenance, fixed eyes, accelerated, full, hard and often wiry pulse, and tenderness when the ribs behind the left elbow are pinched or struck. The same tenderness is noticed particularly in the ox and smaller quadrupeds when pressure or compression is made beside the breast bone. Auscultation over the lower ends of the fifth and sixth ribs and their cartilages, detects a friction or rubbing sound in the early stages, and until liquid has been thrown out into the pericardial sac. This sound may be at first the finest possible creaking, afterward increasing to a distinct rubbing, is synchron-

ous with the beat of the heart, and usually with the first sound. It is distinguished from the friction sound of pleurisy in occurring rhythmically with the sounds of the heart, and not with those of breathing, and from sounds produced in the interior of the heart by its absence when auscultation is made over the carotid or other large artery. This friction sound is lost when serous effusion takes place into the pericardium, but reappears when the liquid is absorbed in the process of recovery. Until effusion takes place the impulse of the heart is strong, often irregular in force, and sometimes accompanied by a purring tremor or, according to Leblanc, a metallic tinkle.

When effusion has taken place, the pulse is weaker and softer, irregular or intermittent, the impulse of the heart is weaker, the friction sound is lost, and the area of dullness corresponding to the heart is increased. Percussion shows it to extend higher than three inches above the breast bone in the horse, and more than two or two and a half inches transversely. It is distinguished from the effusion of pleurisy in this, that the dullness is confined to the anterior part of the chest, having the outline of an inverted cone, and does not extend backward along a horizontal line, and, in solipeds, in not showing equally on both sides. In the smaller animals it may be distinguished by not always occupying the dependent part of the chest when the animal is placed in different positions. As the effusion increases, the heart's sounds, previously strong, become first muffled, then more and more distant until they may become altogether imperceptible. The difficulty and oppression of the breathing increases, the nose is protruded, the eyes more rigidly fixed, and the face more haggard; a venous pulse, apparently due to the compression of the heart and large veins by the fluid, is seen in the lower ends of the jugulars, and the animal obstinately stands as indeed the solipeds do all through the disease. At this advanced stage dropsies of the limbs, sheath, and other dependent parts of the body are frequent.

A painful cough is sometimes, though by no means invariably, present throughout the disease. Emaciation takes place rapidly and in the more acute cases, death ensues in five to eight days. A fatal issue may be delayed until after three weeks, or the affection may merge into a *chronic form*.

In *dogs*, breathing may be moderate at first, during rest, but under exertion becomes rapid, labored, dyspnoëic, the animal standing or sitting with fore legs apart, and elbows turned out, the jugulars stand out full and prominent, and the eyes, mouth and nose are cyanotic. Palpitations and irregularity of rhythm are common; the pulse unequal, becoming small and weak. Standing the patient on his hind limbs restores the lost respiratory murmur and weakened heart sounds in hydrothorax but not in hydro-pericardium.

Chronic Pericarditis is sometimes seen in the *ox* as a result of tuberculosis, and without any preceding acute attack. In other cases it follows the acute. It may be manifested by the local symptoms without the accompanying acute fever. Along with a *slight* fever, there is the oppressed breathing, aggravated by exertion, the weak irregular or intermittent pulse, the muffled or distant heart sounds, the absence of respiratory sounds, and the dullness on percussion over a space represented by an inverted cone at the anterior part of the chest on each side, the venous pulse in the neck and the general tendency to dropsy. (See hydropericardium and adherent pericardium).

If the *pericarditis* has been the result of sharp pointed metallic bodies swallowed and afterwards making their way to the heart, it is sometimes preceded by eructations, tympany, difficulty in swallowing or in rumination, and attended by dropsy under the sternum, but more frequently the heart symptoms are the first to be noticed. It is not usually attended by the high fever of other pericarditis.

Post Mortem Appearances. These do not differ materially from those of pleurisy, to which accordingly the reader is referred. The effusions and false membranes are of course localized in the sac of the pericardium. A frequent termination is a permanent adhesion of the pericardium throughout more or less of its extent to the surface of the heart. In cases of death, the serous effusion is commonly colored with blood, though mostly from a *post mortem* infiltration of blood from the congested lungs. The effusion has been known to measure fifteen liters in the horse. It may be purulent or combined with fetid gases, particularly in traumatic cases. After mild attacks, white patches (milk spots) are often left, extending, it may be only through the pericardium, and in

other cases reaching into the muscular substance. At a less advanced stage the false membranes are yellow, with a rough or villous surface, they may be softened from fatty degeneration or they may be more or less completely calcified.

When the cause has been perforation by a metallic body, it will be found surrounded by exudate enveloping a canal or band extending to the diaphragm or stomach.

Treatment. Pericarditis often proves fatal but it is by no means invariably so in uncomplicated cases. There is especial danger when serous effusion is excessive, when it occurs in a weak and debilitated subject, or when it is complicated by pleurisy, influenza or rheumatism. The preliminary chill may be met by the measures advised for the rigor of pleurisy, but if the malady is developed other treatment is required. The medication is still essentially as for pleurisy, only the primary disease (rheumatism, influenza, pneumonia) must be specially attended to when such is present. Acute pain may be met by carefully graduated doses of opium or aconite and by the moist jacket or fomentations. Some employ ice bags to soothe at once inflammation and pain, and in the absence of rheumatism these may be resorted to. In the small animals leeches may be applied over the cardiac region. Dry cupping is a good alternative applicable to all.

An active purgative is demanded unless the affection is attended by a low type of fever or has occurred during the course of an epizootic disease, (horse, 5 to 7 drachms aloes, cow 1 to 2 lbs. Epsom salts, dog, $\frac{1}{2}$ oz. castor oil). After the walls of the chest have been well fomented they may be enveloped in a large mustard poultice, which must be continued until a considerable effusion has taken place beneath the skin. To moderate and control the heart's action give digitalis (horse and ox $\frac{1}{2}$ drachm, dog, 2 to 4 grains) four times a day. After the purgative has acted, an ounce of nitrate or acetate of potass may be given daily to the larger quadrupeds ($1\frac{1}{2}$ drachms to sheep and pigs, and 20 grains to dogs) in the drinking water. These agents, together with the digitalis, must be pushed to the largest doses when the effusion has taken place abundantly and when it threatens to dangerously interfere with the heart's action. Pilocarpin is a dernier resort, to be used with caution. In similar circum-

stances, ointment or tincture of iodine should be freely applied over the chest in the region of the heart. Mustard and other vesicants repeatedly applied often greatly hasten the reabsorption of the liquids.

From the first the animal must be warmly clothed and every means employed to obtain free circulation and warmth on the surface. The legs must be well rubbed and wound in warm flannel bandages, or this failing, may have mustard freely applied to them. Warm injections must be at the same time thrown into the rectum, and will benefit by soliciting the action of the bowels as well as in raising the temperature of the surface generally. The food allowed should be warm mash of wheat bran, boiled linseed, and similar agents in small quantities.

If the amount of effusion threatens a fatal result, it may be drawn off by a cannula and trochar introduced between the cartilages of the fifth and sixth ribs, by a valvular wound and with antiseptic precaution (see hydrothorax), care being taken to avoid puncturing the heart itself.

The trochar or aspirator needle should be pushed in a direction upward and inward until resistance ceases or it is felt that the heart has been touched. The trochar having been withdrawn, a caoutchouc tube may be attached to it and allowed to depend twelve or eighteen inches, and its lower end should be plunged in a weak solution of boric acid or other antiseptic. This avoids the entrance of air and insures against the introduction of aerial bacteria.

When the vital powers are being exhausted stimulants must be given to support the animal, combined with iodide of potassium. (See advice concerning the allied condition of *Hydrothorax*.)

In the *chronic pericarditis of oxen* the fatality is greater. Treatment consists mainly in counterirritants and powerful diuretics employed in doses determined by the strength of the animal, and combined with stimulants and tonics as in the advanced stages of the acute disease.

For the *dog*, Cadeac recommends tartar emetic ointment rubbed in and closely covered so that the patient cannot lick it. When removed it must be carefully washed off as the dog will readily poison himself by licking the part. It is, however, very

depressing : mustard or ammonia and oil are much safer and better. In high fever he advises acetanilid, 50 centigrams, but if demanded by a dangerously high temperature, it should be stopped as soon as that moderates, lest the heart be depressed unduly. When not imperative because of the high fever, it is better to employ the cooling diuretics, as in pleurisy, and, when the heart weakens, to use digitalis, or strophanthus, or caffeine, in combination with stimulating diuretics like acetate of ammonia (5 grs.), or even sweet spirits of nitre (20 drops).

In complicated forms of pericarditis, attention must be given mainly to the constitutional affection, thus in *influenza* stimulating and supporting treatment is demanded, and in *rheumatism* colchicum, acetate of potass, salicylate of soda, salol and similar agents must be freely administered, though not to the exclusion of counterirritants to the region of the heart, and other measures demanded by the heart diseases.

ADHERENT PERICARDIUM. DRY PERICARDITIS.

Definition. Adhesion of the parietal layer of the pericardium to the visceral, so as to obliterate the pericardial cavity.

Lesions. Causes. This condition is found in all the domestic animals as a result of a pericarditis in which the exudate has been mainly fibrinous and little serous (*dry pericarditis*), or when the serum has been re-absorbed and the false membranes covering the two pericardial layers have united. It may be partial, confining itself to the parts near the base of the organ, where the movement between the two layers is most restricted, or it may be complete, the heart and entire pericardium having coalesced. In partial cases the adhesions may have become stretched so as to form more or less broad bands between the cardiac and mediastinal layers. In other cases there is reason to believe that such partial connections have broken up and become partially absorbed, so that they have left only the thickened white or *milk* spots on the surface of the organ. Other secondary changes are met with. The exudate may extend into the mediastinum in the space between the parietal pericardium and the pleura, where it may form abscess, or a fibroid hyperplasia. In certain cases it becomes the seat of cretaceous deposit so that cardiac calcification starts in this way. In other cases it is the local manifestation of a general disorder. Thus in the *horse*, the adhesion starts in *irregular strangles*, and an abscess forms and finally bursts into the pericardium or pleura. In *cattle* and *dogs* tuberculosis is a more common cause, and tubercles in all stages (red hard nodules, caseated or cretaceous masses) are found in the neoplasm. A cow's heart in the New York State Veterinary College museum is literally embedded in tubercle several inches in thickness. This heart is enormously hypertrophied. *Cancer* is another cause of adhesion in *dogs* and *cattle*. The investing adherent parietal layer, may limit systole and favor dilatation and even hypertrophy, as in the cow's heart just referred to. In other cases, by contracting in process of organization, it compresses the coronary arteries, lessens the blood supply, unfits for exertion, and causes *angina pectoris*, together

with softening and degeneration (fatty, calcic, fibroid) of the heart walls, and it may be, dilatation or contraction of the heart cavities.

Symptoms. The symptoms are in many cases obscure. Changes, amounting to even general calcification, may occur, and yet the function is not incompatible with fairly good health. I have seen the heart of a fat wild duck completely invested with calcareous matter, but the bird seemed, in no way, to have suffered. In cases of calcification of auricle or ventricle in the horse, which is habitually called on for active exertion, the general symptoms of heart disease were manifested under such conditions. Palpitations, irregularities, inequalities and intermissions of the heart beats and pulse, dyspnoea on slight exertion, stocking of the limbs, dropsies, ascites, hydrothorax, venous pulsations, cyanotic membranes, and even muscular cramps in the limbs may be evident. When more or less of these general symptoms follow an acute pericarditis, and persist after the subsidence of fever and local tenderness, or of pericardial effusion, and in the absence of all indications of dilatation, contraction, valvular and other diseases of the heart, adhesion may be suspected. No symptom is pathognomonic, though some have been held to be so. There may be a retraction of the chest wall during systole in cases where the heart is adherent to the wall. This is most marked behind the left elbow. Auscultation may show a perceptible prolongation of the first sound, due to the retarding of the ventricular contractions from firm adhesions to surrounding parts. In partial adhesions this is most likely to be obvious toward the base of the heart, which is usually most extensively adherent. In man a rebound or shock against the ribs has been noticed on the occurrence of diastole, and which is naturally attributed to the dragging of the suddenly released adhesions. A sudden collapse, on the occurrence of diastole, of the previously tense jugular veins, may be referred to the same cause. With complete adhesion the shock of the apex of the heart may be weakened even to extinction. If the impulse is increased higher up, this symptom is all the more suggestive. The gradual weakening of the pulse, even to extinction during inspiration, has been held to be diagnostic, but this often occurs independently, as in the advanced stages of pneumonia in the

horse. Dilatation, contraction, hypertrophy and valvular disease are often present, and are to be diagnosed by the symptoms named under these headings. They are not pathognomonic.

Treatment. Recovery is not to be looked for. All that can be done is to control and steady the heart's action by digitalis or other heart stimulant, keep the patient apart from all excitement, and in the case of meat producing animals, prepare them for the butcher.

TRAUMATIC PERICARDITIS. FOREIGN BODIES IN THE PERICARDIUM.

This may occur in any animal, but is especially common in dairy cows, fed indoors, and liable to swallow pins, needles, wires, nails and other sharp-pointed metallic objects.

Causes. Penetrating wounds from outside may implicate the heart-sac in any animal—fork or bayonet wounds, shot, wounds by horns or tusks,—but in ordinary practice these are so rare that interest centers especially on the foreign bodies which pass from the reticulum to the heart in cattle, and small ruminants. The conditions which predispose ruminants may be shortly stated.

1st. *The gross feeding of cattle and goats.* Cattle naturally take to luxuriant pastures. While sheep, with their delicate and sensitive lips, nibble the grass to its roots, and, prefer the more aromatic products of the hills, cattle require a bite large enough to be drawn in by the prehensile tongue, and thus take to the larger, coarser grasses of the rich bottom land. The cloven bovine foot even is adapted to the soft or marshy ground, expanding as it descends and contracting into smaller compass as it is drawn out. To this is added a ravenous appetite, which has been fostered in domestic cattle to develop the early maturity or rapid fattening, or the phenomenal flow of milk, and the regular periodical feeds favor the same disposition. The bovine animal in domesticity takes a large mouthful and with one or two strokes of the jaws and a speedy insalivation, passes it on into the rumen to be better masticated at leisure in the process of rumination. Hence it is that objects of all kinds and of considerable size are swallowed without the animal being conscious of their presence. In stables, pins and needles, from the clothes of the attendants, drop into the mangers or fodder, nails carelessly handled get into the food, and the cut ends of wires used in *pressing* hay, together with other objects that are almost equally dangerous. While goats do not use the tongue to draw in their food, yet they are notorious for chewing and swallowing non-alimentary substances, so that they too are largely exposed to the danger in hand. Like cattle, and especially pregnant cows,

they will take pleasure in devouring stray articles of clothing with the pins and needles that may be fixed in them. In districts, like Brittany, where cows are habitually cared for by women, this danger is enhanced, and in the vicinity of certain factories, like millinery establishments, wire works, etc., where sharp pointed bodies are scattered, the number of cows perishing from this cause is remarkable.

2d. *The meshes of the reticulum.* The long villi of the rumen and the folds of the manifolds and rennet, yield readily to foreign bodies of all kinds and coming from any direction. But the alveoli within alveoli of the reticulum are calculated to entangle any solid body, and above all any pointed body, and hold it firmly. If any such pointed body once has both ends engaged in two alveoli, they must necessarily impinge with more or less force on the mucous membrane, under the movements of the stomach and its changes of size and form. In such a case the more pointed end will prick more deeply and the constantly repeated wound determines inflammation, ulceration and finally the formation of a track or fistula in which the foreign body lies. The direction will depend on the position of the sharper end, and if that is downward or backward it either fails to do harm, or it opens a track by which the body escapes through the wall of the abdomen or into an intestine, but such a course is rare. When on the other hand the sharp point is directed forward, its active progress is favored by the constant respiratory movements of the diaphragm and the beats of the heart.

3d. The anterior wall of the reticulum is in direct contact with the diaphragm, and the inspiratory contractions of that muscle cause a series of compressions on the sharp point of the foreign body, and hasten the inflammation and absorption. In the next place the convexity of the diaphragm in expiration is such that it comes in direct contact with the pericardium, and thus the shock of the systole is experienced over the point, multiplying the pressures by four or five times the number and keeping up a rapid succession of beats, which determine the progress of the foreign object in the direction from which these impulses are received.

Of other routes sometimes taken by foreign bodies the following may be named :—into the larynx (Pallart) ; œsophagean wall

(Joyeux); wall of the abdomen (Mottet); intercostal region (Cornette).

Lesions. The earliest lesions must be in the mucosa and muscular and serous coats of the reticulum, but these are rarely noticed by themselves. In the second stage there is a fistula passing through the anterior wall of the reticulum and the diaphragm and surrounded by a more or less abundant inflammatory exudate. The thickness of this exudate is in ratio with the size, form and sharpness of the foreign body; a fine, sharp, smooth sewing needle may pass with little irritation or exudate, whereas a coarse, rusty wire or nail, or a coarse piece of metal conveying septic microbes causes much disturbance and an exudate as large, perhaps, as an infant's head. At a still more advanced stage the fistula with its walls of exudate or neoplasm, extends forward through the mediastinum and outer layer of pericardium, or an adhesion having been established between the two layers, the canal extends to or into the heart itself. As far as to the pericardium the fistula is usually very narrow and follows a generally straight course; it may even have narrowed after the foreign body passed through, so that it would not admit of its return. In other cases, however, it remains wide and open and it is evident that the offending body has even receded into the reticulum and disappeared, and several fistulæ have been found side by side no one of them having lead as far as the heart (Boizy). The wall of the fistula may be congested of a deep red; or it may be a seat of infiltration with an abundant serous exudate giving it a yellowish or grayish appearance; or it may be dark in color from pigmentation and hard from fibroid organization in cases of older standing. At intervals it may show one or many abscesses varying in size from a pea, to several inches in diameter, closed or opening into the fistular tract, the pleura or pericardium. The contents of the fistula may be pure, creamy pus, but usually they are dark, frothy and malodorous.

The adjacent pleura is not infrequently implicated, and there may be extensive collections of fluid and false membranes, the liquid being bloody, pink, grayish or translucent, or again purulent, or frothy and foul smelling as in other pleurisies. In the same way the pericardium may be involved and become the seat of extensive false membranes and liquid effusion. Sometimes

the fistula or an abscess may burst into this sac producing purulent or septic infection in the contents. This pericardial liquid may amount to 15 liters (Friedberger and Fröhner,) or even 18 (Trasbot).

Sometimes the lung is implicated having been perforated by the foreign body (Cornette), or involved by extension of infection from the pleura or pericardium to which it has contracted an adhesion. Infiltration and even abscess of the lung is met with.

When the perforating body has become encysted in the pericardium it may remain fixed for months or a year, the walls having been organized (fibroid or calcic).

When the animal survives the perforation of the heart, the latter may show small ulcerous wounds caused by the pricking action of the perforating body as the heart contracts; small, sharp bodies may even penetrate the muscle of the heart and finally the endocardium as well, giving rise to the escape of blood into the fistula, the pericardium, or the fatty tissue. The blood may be in the form of pea-like clots in the connective tissue, or of considerable masses of coagulum. Conversely the microbes reaching the endocardium and blood, attack the less vascular and resistant valves, and lead to exudation, coagula, ulceration and insufficiency.

The exudate in the mediastinum gravitates downward to beneath the sternum and forward beneath the trachea and œsophagus, producing a dropsical appearance of the breast and dewlap.

The mediastinal, bronchial and cardiac lymph glands are always involved, enlarged, congested, reddened and softened.

Symptoms. These vary at the different stages. The earliest manifestations often pass unnoticed, consisting as they do in tardiness and insufficiency of the rumination, capricious appetite and occasional slight tympanies of the rumen after meals. If the gas eructated has a foul odor it is still more suggestive. When the pointed body has been arrested in the gullet and has penetrated its walls, the same disorders of rumination are marked through the formation of a phlegmon and pressure on the vagus nerves.

When the vulnerant body has reached the pericardium the symptoms may become more characteristic, though they vary much according to the nature of the offending body, the infection and the

resulting phlegmonous inflammation. I have seen a milch cow in fine condition, and apparent health enter the prize ring, have the first prize awarded her, and fall dead from a baling wire in the pericardium, before leaving the ring.

In other cases with extensive and microbial implication of the pleura and lung there is a high fever, anorexia, labored breathing and even a swelling with pitting on pressure on each side of the sternum. The exudate in the mediastinum only, is usually too deep and too limited to give satisfactory results in percussion, but the observer may be happy enough to detect a clicking sound from the bursting of bubbles and passing of gas through the fistula. If there is a collection of liquid in the pleura, we may have all the significant symptoms of pleurisy with effusion, and even of hydro-pneumo-thorax.

When the morbid process has reached the pericardium we find as a sequel to the symptoms just named, others that indicate pericarditis, as hurried breathing amounting at times to dyspnoea, readily roused by making the animal walk a few steps. If not accompanied by a moan, when at rest, a grunt is elicited with each step, and the nose is protruded. A pleuritic ridge may show on the flank and expiration is preceded by a sudden closure of the glottis and arrest of the inspiration, and accompanied by a double lifting of the flank. The body and even the neck are stiff and rigid, every movement or step causing suffering and a moan. A cough, short, weak and painful, is now present, roused by any movement, as driving. Palpitations and irregularities, even to intermission of the heart and pulse are to be looked for. Friction sound, synchronous with the heart beat, may be detected, followed later, it may be, by a rushing, splashing or metallic tinkling sound from the presence of both liquid and gas in the pericardium. The sounds are often louder on the right side of the chest than on the left, indicating in such cases, the presence of an abundant exudate on that side of the pericardium. If the patient lies down it is usually on the right side and the difficulty of breathing is greatly aggravated. If not noticed before, dropsical effusion in the dewlap and on each side of the sternum now appears, sometimes only enough to cause pitting on pressure, at others forming a thick oedematous cushion and extending into the fore limbs.

Digestive disorder may be present, shown by liquidity and fœtor of the stools, passage of foul gases, black color of the droppings, periods of constipation alternating with diarrhœa, and impairment of appetite and rumination.

In implication of the myocardium, and endocardium, the functional disorders of the heart are more marked, palpitations, inequality and irregularity of the beats, and intermissions are to be looked for, also blowing heart murmurs from valvular disease or thrombi, and finally venous pulse in the jugulars. The entrance of septic microbes and the formation of heart clots often entail embolism or abscess in distant parts or organs. Or the fistula, opening into the heart cavities, allows an escape of blood into the reticulum and through this into the stomachs and intestines, incidentally admitting hosts of septic microbes into the circulating blood. Gastric and intestinal fermentations ensue, with tympanies and fœtid diarrhœa. In the rare cases in which the patient survives to suffer in this way, it now speedily dies from impaired heart function, pyæmia or septicæmia.

Percussion gives valuable indications in advanced stages. It brings out indications of tenderness and notably grunting with each blow, in ratio with the force of the impact. It exposes a large area of non-resonance, of a conical form, with its base turned upward and backward, and extending from about the middle of the fifth or sixth rib back to the diaphragm near its centre. Sometimes, in advanced stages, the presence of gas in the pericardium may give a drum-like resonance to the antero-superior part of this cone. The conical outline, too, may be modified by a widening out below in case of hydro-pericardium.

Auscultation may show a distant or muffled heart sound, with at times early friction sound, or the later splashing sounds already referred to, but much more characteristically, in given cases, the special sound caused by the passage of gas through the liquid in the fistula. This varies in different cases, with the size of the canal and the abundance of the liquid and gas. It has been variously likened to the sound of *clack-clack*, *glut-glut*, like the sound of water flowing intermittently from an inverted bottle, and especially if into a vessel of sonorous qualities; like the metallic tinkling of drops of water dropping into a vessel half full, or upon a marble table; or even the sound produced by

striking an empty glass with the back of a knife. This sound is synchronous with the heart beat, though it may be intermittent, the intervals representing the periods following the escape of gas and the relief of the tension in the canal. This is one of the most *diagnostic* features of this lesion, and is especially so in stalled cattle, when the attack has appeared and advanced gradually with at first little or no fever; when there have been early gastric disorders; when percussion shows a line of flatness and tenderness from the middle of the diaphragm forward; when there have been functional disorders of the heart; when dyspnoea is produced by slight exertion; and when there is infiltration of the dewlap and sternal region.

Friedberger and Fröhner note a blowing or rushing sound in the large arteries, aortic and pulmonary, when compressed by the exudate, and Cadeac and Brinot a blowing sound synchronous with the contraction of the heart and with expiration, which they attribute to the distension of the pericardium. Finally Bernardin notes the exceptional loudness and clearness of the heart sounds on the left side, behind the scapula, when the exudate is so abundant as to form a specially favorable sound conductor.

Treatment. As a rule treatment is useless, but as the result is almost certainly fatal if left alone, the practitioner would be fully warranted in resorting to desperate surgical measures. In most instances such measures have failed to save the patient. In one case the rumen was opened and a nail, engaged in the wall of the reticulum and in the diaphragm, was withdrawn, with a successful issue (Johow). In a more advanced case the chest wall was opened, opposite the base of the pericardium, on the left side, and the foreign body sought and extracted. The cow recovered and was sold fat, three months later (Bastin). As already noted, very exceptional cases have recovered spontaneously by the slipping of the offending object back into the reticulum through a spacious canal. In others it has taken a new direction and emerged through an intercostal space.

Prevention. This is the only satisfactory resort. The very careful seclusion of nails and sharp-pointed bodies from cow houses, and from barns in which fodder is stored; the disuse of baled hay, or the careful removal of the wires from the bales so

that no small cut or broken portion, can ever reach the rack or manger ; the disuse of ordinary pins and needles about the barn, safety pins being alone used when necessary ; and the avoidance of all sweepings from dressmaking, millinery or tailoring establishments or from any place where pins, needles, or other pointed metallic objects are largely used, will go far toward preventing this deadly affection.

RUPTURE OF THE PERICARDIUM.

This is reported as a rare occurrence in *horse*, *ox*, and *dog*, but the cause in each case has only been conjectured as being a violent blow or shock operating on an already diseased and weakened pericardium. Cuthbert records a case in the *horse* in which the pericardium was lacerated for a distance of eight inches from the apex so as to allow the protrusion of a large part of the heart. The margins of the laceration were inflamed and thickened. Leather publishes a case in which a horse, during work, sustained a laceration of over two inches long. Larcher notes cases of rupture of the root of the aorta, implicating the pericardium, so that a mass of blood escaped into its cavity. In *cattle*, the perforation of the pericardium is commonly due to foreign bodies migrating from the reticulum. Cases of the kind are further charged on perforation by parasites, and vulnerant bodies entering from without. In both *cattle* and *dogs* the presence of tubercles on the pericardium impairs its tenacity and is a frequent cause of perforation. Penetrating wounds are more common in these animals than in horses. Rupture has occurred in septic effusion into the pericardium with the evolution of gas in the cavity. A congenital aperture in the pericardium of a puppy is noted by Paul Bert.

The result of the rupture is shown in sudden and usually urgent phenomena of cardiac disorder and weakness, violent palpitation being associated with an unequal, intermittent, weak pulse gradually becoming imperceptible. Death, usually by syncope, occurs, speedily or after an interval of from one to fourteen days. The lesion is always hopeless.

HYDROPERICARDIUM.

Definition. Dropsy of the pericardial sac.

Causes. This is an effusion occurring, passively, independently of inflammation, and like other œdemas may come from an excess of water in the blood (anæmia, hydrocæmia), or from nephritis, hepatitis, obstruction to the pulmonary or pleural circulation, (pulmonary sclerosis, cancer, tuberculosis, actinomycosis), insufficiency of the left auriculo-ventricular valves, or parasitisms (distomatosis, lung worms, intestinal worms.)

Lesions. The pericardial fluid is considerable in amount, clear or straw colored, and contains a little albumen, and traces of fibrinogen and salts. The paucity of the two latter engredients distinguishes it from the effusion of pericarditis.

Symptoms. These are the objective symptoms of pericarditis with effusion, but without the attendant fever, or the pain on pressure or percussion. It is not preceded by a cardiac friction sound. There are dyspnœa, rapid pulse and breathing, easily roused by slight exertion, a tendency to palpitation, irregular rhythm of the heart, excessive area of flatness on percussion showing a conical outline with the apex below, a low, muffled or distant character of the heart sounds, and it may be a venous pulse in the lower end of the jugulars.

Treatment. Correct the primary disease on which the dropsy is dependent. Then solicit the absorption of the effused liquid by diuretics, digitalis and deobstruents (pilocarpin). Blisters to the region of the heart will often favor absorption, and may be repeated when the effect of the first have ceased. All other measures failing, tapping may be resorted to, but this will be ineffectual while the primary disease-factor persists.

ENDOCARDITIS.

Definition. Pathology and lesions: congestion of the endocardium covering the valves, valves liable through friction and strain, exudation in or on the serosa rendering it opaque, coagula of fibrine on the surface, secondary endocarditis, mycotic, microbial, changes in serosa, distortions and degenerations of valves. Symptoms: as in pericarditis, with violent heart impulse of varying force, irregular, intermittent, absence of local tenderness, no friction sound, no increase in area of dulness, if lesions are in right heart—venous pulse, venous congestion, dropsies. Valve lesions, in mitral valve—general heart symptoms and murmur with 1st heart sound, 2d sound may be repeated and exceptionally a venous pulse—in tricuspid valve—same with constant venous pulse, venous congestion and dropsy; narrowing of the mitral orifice—general heart symptoms and blowing murmur before the 1st sound; narrowing of the tricuspid orifice—same with murmur sometimes audible on the right side; insufficiency of aortic valves—general heart symptoms and murmur with 2d heart sound, double rushing sound in arteries and delay of pulse-beat at jaw; lesions in pulmonary valves—same but without double rush in arteries, or delay of pulse-beat at jaw. Loose coagula. Embolism. Causes: as in pericarditis and strain on valves, and poisons and microbes in the blood. Prognosis grave. Treatment: as in the early stages of pericarditis, antirheumatics and germicides more, and diuretics less desirable. For clots iodides, alkalies.

Definition. Inflammation of the serous membrane lining the chambers and covering the valves of the heart.

Pathology and Morbid Anatomy. Nearly all cases become complicated by microbial invasion and this infection may be considered as the essential factor in the disease, yet accessory and predisposing causes are hardly less important, as on these depend the susceptibility which renders the microbial invasion effective. The accessory causes and symptoms will be better understood after the diseased conditions have been comprehended. The earliest changes are the reddening and thickening of the lining membrane of the heart but above all, of that covering the valves. The valves are particularly exposed to inflammation by reason of the friction of the blood when violently forced through the narrow opening in excited conditions of the heart, by the strain thrown upon them from the violent contractions of the heart, or the recoil of blood in the arteries, and by their susceptibility in common with all other fibrous structures to rheumatic inflammation. The redness is of the ramified or branching kind, charac-

teristic of inflammation, and is neither removable by washing the surface nor does it correspond in position with the colored portion only of a clot, which the cavity in question may contain, as seen in bloodstaining occurring after death.

There is further exudation of plastic lymph into and beneath the serous membrane, rendering it opaque, white and thick, or on its surface forming granular elevations, and in the case of the valves becoming moulded into ridges or festoons by the mutual pressure of the different flaps on each other. The inflamed surfaces are further liable to be covered by masses of blood clot in successive layers, deposited by the action of the fibrinogenous matter developed in the inflamed part. These clots sometimes accumulate in considerable masses, firmly adherent to the heart's walls or valves by their attached surface, but soft and filamentous on their free aspect. These clots or polypi, as they have been called, are soft and loose on their free surface, and become firmer toward their points of attachment. In other words their consistency is in direct ratio to their age. If of old standing they are usually pale yellow or white and streaked with red, while if recent they are mostly red throughout. They vary in size from a thin film to a mass filling up nearly the entire cavity in which they are lodged, and as they frequently extend through the auriculo-ventricular openings or become applied against this or the opening of the great artery, they seriously and sometimes fatally interfere with the circulation. Leblanc asserts that large masses of this kind may be deposited in a few days or even hours, causing sudden deaths, and especially in dogs. He has held that other circumstances than endocarditis cause these fibrinous deposits, and especially the absorption of pus, or the sudden suppression of a long standing discharge, as in catarrh of the air-passages. If death does not immediately ensue, these fibrinous deposits may become vascular, as is the case with false membranes on the pleuræ, becoming organized into fibrous tissue, or even degenerating into calcareous matter, necrotic debris, or pus, several instances of which as occurring in horse and cow are on record. The microbes of endocarditis may attack the valves *primarily*, or these latter may suffer from a *secondary* invasion by microbes that have already formed colonies in some other parts of the body. Very frequently in animals, as in man, the invasion of the serosa

covering the fibrous valves is but an invasion by the germs of rheumatism which have been already colonized in joints, fascia, tendons or other fibrous structures in other parts of the body. That it should concentrate its assault on the auriculo-ventricular and arterial valves, is doubtless explained by the stronger blood tension operating on these than on the other valves in the circulatory system,—in the veins, for example, and the lymphatics. This view is corroborated by the fact that the valves on the left side of the heart, that are subjected to the greatest blood tension, are the most subject to attack in extra-uterine life, while the right side which contracts less powerfully, causing less tension, is less often affected. This is the more remarkable in view of the fact, that the right side is by far the more frequently affected with dilatation, the weaker walls being unable to compress the blood as forcibly, and their readiness to yield to pressure further restricting the strain that might be thrown on the valves. The notorious influence of cold (rain, sleet, snow, wading or swimming in cold water, cold draughts, etc.) in laying the system open to rheumatism is equally operative in predisposing the cardiac valves to the specific rheumatic attack. As in articular rheumatism, too, a traumatic or mechanical lesion of a joint will predispose it to rheumatism (the essential microbian cause being present) so, in the cardiac valves, their strain or injury lays them more open to attack.

Again, morbid conditions of the circulating fluids, modifying the nutrition and other conditions of the tissues, tend to predispose these (valves) to colonization by the microbes (of rheumatism, etc.,) that may be present.

But, although endocarditis is very often rheumatic, in a debilitated condition of the valves, it may be the result of almost any microbian infection then present and generally diffused through the blood. In the *horse*, it is especially found in connection with omphalitis, pneumonia, pleurisy, influenza, arthritis, abscess, pyæmia, osteomyelitis, etc. The larva of the sclerostoma equinum is an occasional cause. In *cattle*, aside from the common rheumatic factor, it is especially connected with tuberculosis, omphalitis, arthritis, bone disease, pleuro-pneumonia, pyæmia, etc. In *swine*, besides the common factor rheumatism, tuberculosis is a frequent cause, also bone and joint diseases, and the in-

fectious fevers, especially rouget (erysipelas), and in this connection, omphalitis, pyæmia, gangrenous and diphtheritic infections. etc. In *dogs*, we must bear in mind the special predisposition to heart disease, also to rheumatism and tuberculosis which are both liable to attack the heart. Hunting dogs are especially liable through the great strain thrown on the valves. Again, omphalitis; bone diseases; parturient phlebitis; distemper; infected wounds, operative and accidental; infective tumors of the digits and elsewhere; and ulcerous stomatitis have each been the starting point and source of infection in endocarditis. Hæmatozoa must not be overlooked; strongylus vasorum and filaria immitis, living in the heart blood, are especially injurious. In a recent case of filaria immitis, in a pointer, from Virginia, with forty mature worms in the dilated right ventricle and in large aneurisms on the right pulmonary artery, the blood meanwhile swarming with embryos (1 per cubic millimetre), the left flap of the tricuspid valve was represented by an uneven ridge of vegetations only, and the few remnants of its cordæ tendinæ were short and flattened down against the septum ventriculorum.

In the domestic animals generally, as in man, the heart valves are a favorite seat of different microbial infections of very varied kinds. Anærobic organisms are, in the main, debarred on account of the oxygen in the blood, and the left heart, with its more highly oxygenated blood is the most common seat of the infection. Lion claims that in foetal puppies, ulceration of the valves is more common on the right side, which in intrauterine life receives the most highly oxygenated blood.

In the early stages there may be a mere swelling of the valves, with as yet a smooth, unbroken surface, but with enlargement and increase of the connective tissue cells, later fungous vegetation starts out from the surface, and on these fibrine of the blood is deposited in layers.

Besides the formation of clots on their surfaces other changes occur on the cardiac valves as the result of inflammation. The organization of the exuded lymph within and upon them leads to rigidity, loss of elasticity, unevenness of their surface, contraction and puckering so that they can no longer approximate to each other, but leave the orifice imperfectly closed. They may,

moreover, have gristle or earthy salts deposited in their substance. The osseous degeneration of such new products, appears to be the most common cause of those ossifications of the heart, of which specimens are to be found in nearly all veterinary museums.

Chronic valve disease is thus found to be a common result of endocarditis, and from the obstacle presented to the flow of blood through the different cardiac orifices by the rigid, inelastic and distorted valves, hypertrophy of the heart frequently supervenes.

In our domestic quadrupeds ante-mortem clots and fibrinous polypi have been chiefly formed in the right side of the heart, and diseased valves in the left.

Horse. Symptoms. The general symptoms agree in many respects with those of *pericarditis*. There are the same general symptoms of fever (temperature 101° to 106°), the same pinched, anxious countenance, the same shortness of breath and oppression when moved, the same violent heart's action, and the same rapid excitable pulse, tending to be irregular and intermittent. The dyspnoea may come on in paroxysms, sometimes without apparent occasion, the breathing becoming short, quick and panting, too quick to be counted, the head and neck extended, the nostrils and chest walls dilated, and the mucosæ deeply congested. In some cases epistaxis has supervened and in others laryngeal paresis,—stertor. Among the more specific symptoms are a very violent impulse of the heart against the left side, varying in force, however, in successive beats, a metallic tinkling accompanying the impulse, and sometimes heard at some little distance from the body, a blowing murmur as soon as the changes in the valves render them insufficient to close the orifices, and, if the obstruction exists on the right side, venous pulse, general venous congestion, and dropsical swellings.

The hand, applied flat on the ribs just behind the left elbow, detects the violent impulse of the heart, and often also a fremitus or thrill, which has been compared to the purring of a cat.

The pulse may at first have considerable force, but as insufficiency of the valves ensues, it becomes small and weak, its weakness forming a most marked contrast to the violence of the heart's impulse against the side. The irregularity and intermission of

the pulse is to be ascribed at first to the impaired nervous energy of the heart, though later it is often due to the obstacle presented by clots to the flow of blood from the heart, so that a beat sometimes takes place without a corresponding pulsation. It may reach 80 or 160 per minute in horse or ox. Syncope sometimes occurs.

The blowing murmur when heard is one of the most characteristic symptoms, but must be carefully distinguished from other allied heart sounds. If very loud it may be confounded with the friction sound of pericarditis, but may be differentiated by its invariable coincidence with some particular portion of the heart's beat. The absence of local tenderness is another distinctive symptom. Again in pericarditis, effusion takes place early, annulling friction sound, and diminishing alike the impulse and the sounds of the heart.

It is of less practical value to be able to distinguish the precise seat of the murmur, yet the following data will guide to such a conclusion.

Simple induration or insufficiency of the Left Auriculo-ventricular (Mitral) valve. Paroxysms of palpitation, oppression, and difficulty of breathing; vertigo with loss of control over the limbs and vacillating gait; stupor, coma; slight tremor and blowing noise with the first sound of the heart; heart's impulse violent, but irregular in force, sometimes double; pulse feeble, irregular, unequal or intermittent; sometimes, though not at all constantly, a venous pulse in the lower end of the jugulars. In chronic induration of this valve, or in osseous or cartilaginous degeneration, the same symptoms are shown. The more general symptoms may, however, require exercise to develop them.

Induration, etc., of the Right Auriculo-Ventricular (tricuspid) valve. The symptoms are almost identical with the last. Venous pulse is constant, and, particularly after exertion, the veins generally are distended. Dropsies are more common.

Narrowing of the Mitral orifice. In addition to the same general symptoms as the last named lesions, there is a sighing, blowing, purring or rasping sound, according to the degree of narrowing, heard *before* the first sound of the heart. It is the noise of the blood rushing through the narrowed orifice between auricle and ventricle. It is usually loudest behind the middle of

the shoulder on the left side. Feeble pulse, frequent imminence of suffocation, and filling of the limbs, etc., are nearly constant.

Narrowing of the Tricuspid orifice. Symptoms nearly identical with the last. Venous pulse more constant. Blowing murmur sometimes loudest on the *right* side of the chest.

Induration or insufficiency of the aortic valves. Blowing murmur with the second sound of the heart. Double rushing sound in the carotid with each heart's beat. There is an appreciable interval between the beat of the heart and corresponding pulsation at the jaw.

Induration or insufficiency of the pulmonary valves. Blowing murmur with the second heart sound, but no corresponding double sound in the carotid, nor any marked retarding of the pulse.

Loose coagula in the heart, or adherent ones (*polypi*), produce one or other of the above class of symptoms, according to the particular orifice they tend to block, or the valves the function of which they impair.

Anæmia and leukæmia may have blowing murmurs with the first or second heart sound.

Embolism. Plugging of arteries. Another class of symptoms sometimes supervenes because of loose clots being washed on into the arteries, and blocking them when they reach those that are too small to transmit them. These symptoms will be as varied as the organs the arteries of which are plugged. If in the brain, there may be dullness, stupor, vertigo, somnolence, delirium; if in the liver, biliary and digestive derangement; if in the lungs, cough with the other signs of pneumonia and abscess; and if in the limbs, lameness and paralysis, (brought on or aggravated by exercise, and often removed by a few minutes' rest), wasting of the muscles, etc. (*See Embolism*).

Cattle. Symptoms. The general and physical phenomena of the disease are essentially the same as in the horse. The primary disease of which this is a sequence, is, however, liable to differ, and attention to this may assist in diagnosis. Thus a preceding rheumatism, tuberculosis, or metritis, should be especially looked for, without forgetting the diseases of the liver, kidneys, navel, bones or joints, which may be the cause of the heart disease. The shock of the heart's impulse is sometimes excessive and the

sound loud, and the morbid murmur may take on a purring or humming character. In cattle, as well as in the dog, it is possible to auscultate the two sides of the heart separately and thereby to identify the exact seat of the murmur.

Pig. Symptoms. Following an infectious disease, or constituting a form or complication of it, endocarditis is necessarily associated with all the symptoms of such affection. In the common specific fevers, discoloration to cyanosis of the skin of the white pig and its visible mucosæ is to be looked for. Cyanosis is, however, likely to appear to an excessive degree, even independently of these diseases, whenever the diseased valves have become insufficient. There may have been previous symptoms of rheumatism, or bone and joint disease, or of omphalitis. The diagnostic symptoms are, however, those of the heart, violent impulse, palpitations, fremitus, intermissions, irregularities, and, above all, the blowing or purring murmurs with the first or second sound.

Dog. Symptoms. In this animal the primary disease is likely to have been distemper, rheumatism, tuberculosis, bone disease, or ulcerous stomatitis, and the supervention of morbid heart sounds which do not become weakened by effusion as in pericarditis, may well suggest endocarditis. A dry paroxysmal abortive cough, greatly accelerated pulse (130 to 190), difficult breathing, dyspnoic on slight exertion, palpitations, inequalities in rhythm, intermissions, strong heart-shock, and a fremitus conveyed to the hand which grasps the sternum, are to be looked on as indicative of endocarditis. As auscultated over the breast bone the sounds are remarkably clear and the blowing or purring murmurs with the first or second heart sound can not only be clearly heard, but may be distinctly referred to the affected side of the heart and to the particular valve, auriculo-ventricular or arterial. Mathis says that a blowing murmur heard on the right side over the fourth rib, the patient's nose being held so as to suppress all respiratory sounds, is an infallible sign of endocarditis.

Diagnosis from pericarditis is determined by the absence of the early friction sound, systolic and diastolic, and of the later distant, muffled heart-sounds after the friction sound has ceased; also by the absence of tenderness on manipulation or percussion, and by the presence of the valvular, blowing or purring sounds.

From *myocarditis* it is distinguished by the greater force of the heart impulse and the pulse, by the greater loudness and clearness of the heart sounds, by the diminished tendency to faintness and syncope and above all by the onset of blowing valvular murmurs and the lack of duplication of the heart sounds (arrhythmia, polycratic heart).

From *pleurisy* it is differentiated by the absence of respiratory friction sound, of the short, painful, hacking cough, the pleuritic ridge, intercostal tenderness, and later of the flatness in the lower part of the chest corresponding to the effusion. The forcible impulse and loud sounds of the heart in endocarditis, the faintness or fainting and the supervention of valvular murmurs are further diagnostic signs.

From *pneumonia* it is easily distinguished by its own characteristic signs, as well as by the absence of definite areas of flatness on percussion, with zones of crepitation around them. The early rusty discharge and the later muco-purulent one are absent in endocarditis.

From *influenza* it is to be distinguished by its virtual sporadic appearance, without any evidence of the rapidly extending qualities of a plague; by the marked excess of the cardiac movements and sounds; and above all by the valvular murmurs.

Causes. These are in the main the same as those of *pericarditis*. Microbian infection is the main cause as already stated under pathology. Weak health, exposure to extremes of weather, punctures with foreign bodies, but above all, primary infected centres elsewhere, including the rheumatic condition are common causes. Indeed rheumatism appears more prone to attack the serous membrane lining the heart cavities than that enveloping it externally. One reason for this is to be found in the great and incessantly recurring strain on the fibrous structure of the valves, and particularly in hard-worked horses and hunting dogs in which the strain is often extreme. It has been argued that the increased blood pressure caused by digitalis is an appreciable cause. Its frequent connection with rheumatism is shown in the rheumatic lesions of joints and fibrous structures seen in carcasses dead of endocarditis.

Diseases in the muscular substance of the heart, as cysts, abscess, etc., frequently extend to the endocardium.

Among other causes must be mentioned disease-changes in the blood. These may act on the valves directly, as in the case of lactic acid injected, by Dr. Richardson, into the peritoneum with the view of producing rheumatism, and successfully as regards the lesions of the cardiac valves; or indirectly by determining coagulation and irritation of the lining membrane coming into contact with the clot. The very fibrinous and plastic state of the blood in extensive inflammations is a probable cause of the occurrence of clots in the heart, and the frequency of such clots in the dog has been ascribed to the plasticity of his blood (Leblanc). The injection of pus into the blood or the absorption of microbes from diseased surfaces will sometimes produce ulcerative disease of the valves. The same is true as regards the germs of omphalitis, pneumonia, arthritis and other infectious diseases.

Lafosse records certain cases of endocarditis due to extension of the disease from inflamed veins.

Prognosis. Endocarditis is always attended with great danger to life, but it is more likely to terminate in chronic valvular disease which quite unfits the animal for useful work. Mild cases may terminate in complete recovery.

Treatment. This is in the main the same as that adopted in the early stages of pericarditis. Absolute rest is of prime importance. Laxatives, sedatives and counterirritants are to be mainly relied upon. Acetanilid will reduce excess of temperature but endangers heart failure. Belladonna and chloroform on the chest behind the left elbow may be used. As there is not the same danger from effusion, diuretics need not be pushed to the same extent. Digitalis must be avoided if possible until the high fever subsides. This applies equally to strophanthus, convallaria and other heart tonics. In infective cases quinia, salicylate of soda, salol, or hyposulphite of soda may be given. Later give tincture of muriate of iron.

In rheumatic cases, treat as for an acute attack of rheumatism. Frequent large doses of salicin, salicylate of soda, salol, large doses of acetate of potass and colchicum, warm clothing, and counter-irritants to the region of the heart are especially demanded. (See Rheumatism.)

When clots are suspected, and when endocarditis threatens to

lapse into the chronic form, it is recommended to give iodide of potassium (horse and ox 1 drachm, dog 5 grains twice or thrice daily) with carbonate of ammonia or of potass and bitter tonics. A lengthened rest after apparent recovery is essential to avoid permanent valve lesions.

In case of meat producing animals (cattle, swine) it is usually desirable to turn them over to the butcher at the earliest possible moment. This is especially so when the patient is likely to retain and diffuse the microbes of a pestilential disease like one of the swine fevers.

CARDITIS. MYOCARDITIS.

Definition. Rare. Complicates pericarditis and endocarditis, wounds of the heart, and tubercular and other deposits. **Symptoms.** **Treatment.**

Definition. Inflammation of the muscular substance of the heart.

This is a comparatively rare affection and is necessarily limited to a small portion of the heart's substance, otherwise the cardiac contractions must cease in obedience to the general law that the normal function of an inflamed organ is for the time abolished. It is mainly seen as a concomitant of endocarditis or pericarditis, and extends only to the superficial muscular layers; or it results from a wound, as in the penetration of the heart by a needle or other sharp-pointed body, and is then equally circumscribed. It has been seen as a complication in infectious diseases—apthous fever, influenza, infectious bronchitis, strangles, pyæmia, septicæmia, pneumonia, in cows, parturient metritis and tuberculosis, in dogs, distemper.

The evidences of the existence of carditis are chiefly the lesions met with after death. 1st, The existence of abscesses in the heart's substance associated with polypus (Gowing, Leblanc, etc.) or otherwise (Reynal). Also diffuse suppuration in the heart's substance (Puze, etc.) 2nd, Softening of the muscular substance, a state occasionally met with when an animal has died of ruptured heart. This is shown in spots and patches of a yellowish red or grayish red color, soft and friable, fibrillation largely effaced, but with cloudy swelling and cell infiltration. It may however be mainly a degeneration into fatty granules. 3rd, Ulceration of the walls of the heart, as reported by Mercier in a case of endocarditis. 4th, Transformation, and induration of the heart's substance whether into fibrous tissue, cartilage or bone. This last condition of the walls of the right auricle and ventricle has been repeatedly seen in old horses, the change being in certain cases so extensive that one is left in wonder as to how circulation could have been carried on. Three specimens of this kind were preserved in the museum of the Alfort Veterinary College, Paris, and the Royal Veterinary College,

London. Lafosse records two cases of gangrene of the internal layers of muscle in endocarditis.

The *symptoms* are those of acute heart disease, generally modified somewhat by the precise location of the inflamed spot, and *treatment* need not differ materially from that applied for inflammation of the investing membranes, inner and outer, and for the infectious disease which it complicates. Calming of the heart action by digitalis, strophanthus or sparteine may be useful, and eliminative.

CHRONIC VALVULAR DISEASE OF THE HEART.

This, as already noted, is a common result of endocarditis, the valves being most obnoxious to disease in such cases. The *symptoms* are those mentioned under endocarditis as characterizing diseases of the different valves, such as incapacity for exertion, difficult breathing, palpitation, irregularity or intermission of pulse, venous pulse, abnormal heart sounds, unsteadiness of the limbs when driven, and dropsical swellings in the limbs and elsewhere. The reader is referred to endocarditis for particulars, it being borne in mind that these symptoms are not in this case associated with fever.

Horses affected in this way are useless. Cattle may sometimes be partially fattened by preserving them from all sources of excitement, by keeping the bowels regular and by combatting any paroxysms with sedatives, such as aconite, veratrum, hydrocyanic acid, or opium, and with digitalis.

FATTY DEGENERATION OF THE HEART.

Causes : improvement in the direction of easy fattening, inactive life, best breeds of butcher cattle and pigs suffer. Symptoms : weak, irregular, intermittent pulse, palpitation, unfitness for exertion, general heart symptoms.

In addition to the fibrous and bony transformations to which the substance of the heart's walls is subject, a fatty metamorphosis is frequently met with. In most cases the fat accumulates in great masses externally (*cor adiposum*), but in others the muscular tissue has to a greater or less extent lost its natural structure and fatty granules have taken the place of the sarcous elements (fatty degeneration). In overfed oxen, sheep and swine, the right cavities of the heart rarely escape dilation, and this condition is very often accompanied by the fatty change. Virchow has shown that high-bred English pigs imported into Germany are subject to a similar affection of the entire muscular system. It may occur during wasting diseases and from phosphorus, lead, antimonial or arsenical poisoning.

In *solipeds*, it occurs in connection with maladies which interfere with the blood supply to the cardiac walls, as in pericarditis, atheromatous or other contraction of the coronary arteries, pulmonary emphysema, hydropericardium ; or that interfere with the metabolic changes in the organ (strangles, influenza, contagious pneumonia.)

In *dogs*, it follows distemper, pericarditis, compression or narrowing of the coronary arteries, tuberculosis, emphysema, and in old, pampered, overfed animals.

The *symptoms* are weak, irregular and intermitting pulse, palpitation on excitement, weakness of the heart's impulse in the intervals, incapacity for exertion, sighing, Cheyne-Stokes respiration, loss of control over the limbs when hurriedly driven, and tendency to dropsy. It is often associated with dilatation, is rarely distinguishable from it in life, and is equally beyond remedial measures. The feeding animals most commonly affected can usually be fattened if removed from all sources of excitement. In case of phosphorous, antimonial or arsenical poisoning, improvement takes place when the poison is stopped.

In dyspnoëic attacks, relief may be had from the inhalation of amyle nitrite, and the tone of the heart may be improved by *nux vomica*, *caffeine* or *convallaria*.

CALCIFICATION OF THE HEART.

Calcification of the cardiac walls and especially of the auricles, has been met with a number of times in the horse and ox, yet strangely enough, there is no record of it in the dog, though that animal is often allowed to live out his days, and is, moreover, especially exposed to heart strain and heart disease. That small animals are not exempt is shown in the remarkable case of a wild duck quoted under *dilatation*.

Causes. It has been attributed to old age, glanders, endocarditis and pericarditis, to which may be added dilatation. The primary change is usually the formation of fibrous tissues by the organization of an exudate. The continuous strain on this organizing exudate in connection with dilatation, appears to be a stimulus to the calcareous deposit, which is especially common in the thin, yielding walls in the right heart. The affected auricle may be several times its natural capacity and the terminal ends of the veins may show a sacular distension, yet it is rare to find a calcification of the venous walls. To this the thin, yielding wall of the asygos sometimes makes an exception.

Lesions. The calcified centres are sometimes in isolated plates, from a millimeter to several centimeters in diameter, and other times the whole roof and walls of the right auricle are involved. Even with extensive calcification the different plates often remain united by intermediate fibrous material, so that the normal contractions of the heart can still, in some measure continue. The plates are red, hard, resistant, and crackle under the knife, about as does the cancellated tissue of bone. Toward the margins they are paler and devoid of lime salts, resembling the foetal cartilage of ossifying bone. Under the microscope are found the large, round cartilage cells, merging into the stellate osteoblasts, but scattered irregularly through interweaving bundles of white, fibrous tissue in place of being arranged in Haversian systems.

Symptoms. In some cases no symptoms, even of heart disease, have been observed ; horses have gone about ordinary, moderate work without manifest inconvenience, just as the fat wild duck, mentioned under *dilatation*, seemed to have lived and prospered, in spite of its somewhat precarious, hunted existence. In other

cases there have been symptoms of dilatation such as increased area of dullness ; or of insufficiency of the valves, as in blowing murmurs ; also such general signs of heart disease as dyspnœa, palpitations, accelerated, irregular or intermittent pulse on exertion, venous pulsation in the jugulars, faintness, early weariness, sluggishness, stocking of the hind or fore legs, œdema of the sheath or under the chest and abdomen, and sudden death by syncope. Hughes has noted night aggravations in certain cases, perhaps by reason of the lack of fresh air.

Treatment is manifestly hopeless.

NEW FORMATIONS IN THE HEART. TUMORS. PARASITES.

Glanders, abscess, cancer, epithelioma, sarcoma, melanosis, myxoma, myoma, tubercle, polypus, nevus, parasites—echinococcus, cysticercus, tenuicollis, cysticercus cellulosa, trichina, sarcocyst, filaria immitis, strongylus subulatus, strongylus vasorum.

1st. **Deposits of Glanders.** In many cases of glanders and farcy in horses the specific product is deposited in the heart as well as in other internal organs. Such deposits are small but numerous, infiltrating the muscular tissue; their cut surface is dry, finely granular and of a yellowish white color.

2d. **Abscesses** are sometimes formed in the heart from the colonization of microbes from suppurating surfaces.

3d. **Cancer of the heart** has been noticed chiefly in dogs by Leblanc and Cadiot. It occurs only consecutively to cancer in other parts of the body, yet it has sometimes acquired considerable dimensions (2 fists Cadiot) and interfered materially with the movements of the heart.

4th. **Epithelioma.** In a case of generalized epithelioma in an old *mare* the heart was involved (Morot); in another case the pleura were involved with a mass as large as a hen's egg in the right auricle, and others like peas in the ventricle (Weber and Barrier).

5th. **Sarcoma** or small celled tumor is common in the heart of *horse* and *dog*. In the *horse* this has been associated with sarcoma of other viscera, and of the muscular system (Morot), or isolated on the valves of the left heart (Percivall), or apex (Page). In the *dog* it has been found in the ventricle, left (Bournay), right, (Cadeac).

6th. **Melanosis** of the heart has been repeatedly noticed in the horse. Some if not all such cases should be classed with cancers, as these internal deposits of black coloring matter in solipeds, have, in our experience, mostly possessed malignant characters, though they are usually simple tumors as developed singly in the skin of the horse. These black masses usually project beneath the pericardium or endocardium.

7th. **Myxoma** has been found in the *horse*, in the septum ventriculorum (Koch), in the right ventricle (Lamberlucchi) and

in the left ventricle (Essmann). In *cattle* this forms polypoid masses in the vena cava, and right auricle (Yemgers, Blamberg, Hess, Gamgee) and in the right ventricle (Guimberteau, Germain). These are irregular in outline, sometimes pediculated, of variable color, a yellow, gray or black, and with a nucleus of a gummy, old cheese, or lardaceous consistence.

8th. **Myoma.** Husson records a case of this kind in a two-year-old *colt*, the fleshy excrescences growing from the tricuspid valve. Jungers found a polypoid, pediculated myoma in the auricle of a *dog*. The general composition was of elastic and muscular fibres.

9th. **Cysts** with serous contents were found in the cardiac septum of two cows which had died suddenly (Lambreaux).

10th. **Tuberculous deposits** have been met with in the substance of the heart in cases in which the lungs or other organ were the seat of this disease.

11th. The *fibrous growths* or *polypi* due to the deposition and organization of fibrinous material from the blood have been referred to under *endocarditis*.

12th. Gamgee reports the existence of a vascular tumor of the right ventricle of a horse in the museum of the Turin Veterinary School. It consisted of varicose veins ramifying beneath the endocardium which in its turn was healthy.

13th. The parasites found in the heart are various. *a.* One, the **Echinococcus Veterinorum**, has been repeatedly found in the substance of the heart or projecting from its inner or outer surface. *b.* Another, the **cysticercus tenuicollis**, has been met with in the pericardical sac of a calf (Reed). *c.* A third, the **cysticercus cellulosa** invests the muscular structure of the heart of measly pigs, and the **cysticercus bovis** that of calves. *d.* The heart, like other voluntary muscles of hogs, occasionally contains *trichina spiralis*. *e.* Rainey's cysts (**sarcocysts**) are microscopic ovoid bodies usually found in the hearts of oxen and other animals. *f.* A round worm, **filaria immitis**, first described as *filaria papillosa hæmatica* by Delafond and Gruby, lives in the blood of the dog, is one millimeter thick by fifteen to forty centimeters long. It may obstruct the pulmonary artery (Serres, Law), or the mitral orifice (Silvestre). It may cause various nervous and dropsical disorders, and even sudden death.

Its mode of entrance is unknown. *g. Strongylus Subulatus*, 1 to 2 mm. long by 70 to 90 μ . in thickness was found in numbers in a nodule of a dog's lung, and the dorsal vein of the penis of a dog (Leisering). *h. Strongylus Vasorum* in the right auricle and ventricle of dogs, in pea-like blood clots. It is 14 to 21 mm. long by 1 m. in thickness (Serres).

-RUPTURE OF THE HEART.

In the lower animals ruptures of the heart have been observed as the result of (a) extraordinary exertion, (b) violent concussion, and (c) ulceration and degeneration. The rupture of the fatty heart in the lower animals is not common.

Rupture during severe exertion occurs in the perfectly healthy heart. The ruptures take place in the weakest point, and most commonly in the fibrous ring which encircles the base of the heart and attaches the great aorta. This is occasionally seen to happen in very spirited horses during a severely contested race or when a heavy load is being dragged up hill. Percivall mentions the case of a horse at a Woolwich racing meeting, which had just lost a heat by half a head and which died just after passing the winning post, with ruptured right auricle. Stenosis of the aorta predisposes to rupture of the left ventricle, (Cadeac).

Cases occur during coitus (Hering), tympany (Anacker, Mayer, Perdan) and operations (Stockfleth).

Rupture from Concussion more frequently implicates the muscular walls which have not the same power to resist a blow when in a relaxed condition. Parker met with a case of rupture of the right auricle at its base or at the line of its union with the ventricle. The subject was a pony which ran away down hill and struck his right shoulder violently against a cart wheel. In other instances the rupture takes place in the posterior vena cava, and particularly if its walls have been the seat of disease. Gamgee found rupture of the commencement of the azygos vein in oxen killed by pithing, in the slaughter houses of Ferrara, and Professor Maffei subsequently found that out of 3,095 oxen killed in these abattoirs, 57 had this vein ruptured. Gamgee's explanation of the occurrence is that "the instant the animals are pithed,

the walls and contents of the chest become paralyzed, the heart becomes an inert bag filled with fluid, the jerk of which as the animal falls, causes rupture of the containing vessel at its weakest part, and this is in truth the vena azygos, whose walls are thin and only protected externally by the pleura." Hertwig gives other cases resulting from falls.

Perforation of the heart from ulceration is sometimes seen in cows when sharp-pointed metallic bodies from the stomach make their way into its substance. An alleged case of rupture following ulceration of the walls of the right ventricle is recorded by Gaultet.

Inflammation, softening, fatty and calcareous degeneration, dilatation, atheroma, and the presence of parasites in its substance, render the heart more friable and predispose it to rupture.

Lesion. The rupture during systole is often at the fibrous ring encircling the aorta or pulmonary artery; in other cases from concussion, in the muscular wall of ventricle or auricle. The following seats have been noted: auricles, fibrous rings at the base of the auricle, or encircling the aorta or pulmonary artery, through the body or near the apex of the left ventricle, at the azygos. The laceration in the muscular wall is usually in a line with the fibres, and from a third to an inch in length. The outer edges of the wound are much more irregular than the inner. The pericardium is filled with blood, and the muscular fibres on each side of the opening are torn and shreddy and filled with blood clots. The blood has been known to escape into the pleural cavity and in case of a foreign body coming from the reticulum, it may pass through the fistula into the stomach.

Symptoms. Death may be practically instantaneous. If delayed there is hurried breathing, anxiety, weakness, pallor of the mucous membranes, staggering, trembling, vertigo, stupor, and convulsions.

In *dogs*, rupture of the heart has been noted in connection with operation for ascites (Stockfleth, Benz), with purpura hæmorrhagica (Mathis), with a fall of 20 feet (Rodet), with arrow-wounds and other traumas (Nocard), also with ulcerous endocarditis (Mathis.)

In a *pig* Rosario saw a single case in a pale heart and anæmic animal.

In *chickens* Larcher records two cases, and Cadeac two more associated with diphtheria.

DISEASES OF ARTERIES.

The chief morbid conditions seen in arteries are : wounds, inflammation, thrombosis, embolism, degeneration and aneurisms. Wounds belong essentially to surgery.

ARTERITIS. EMBOLISM.

Internal and external arteritis. Thrombosis, from inflammation. Embolism. Bruising. Stretching. Ligature. Lesions. Extension of clot, color, consistency; adhesion, lamination. Composition of clot. Condition of vessel. Changes in muscles. Causes : muscular tension, embolism. Heart clots, venous clots. Infecting debris. Symptoms : paresis or paralysis on exertion, local suffering, tenderness, firm, swollen artery, derangement of nutrition and function, atrophy. Chronic arteritis : Atheroma. Changes in serosa. Fibrous thickening, atrophy, dilatation. Sclerostomes. Treatment : rest, anodynes, alkalies, massage.

Inflammation of arteries has been divided into *external* and *internal* arteritis, according as it affects the fibrous sheath of the artery or its inner lining membrane.

In *external arteritis* the exudation of lymph often forms a protecting layer around the vessel, while the inner coats continuing sound, the current of blood remains unimpaired. Even when suppuration takes place in the vicinity of a large artery, that vessel may pass through the center of the abscess and convey the blood as freely as before. The nutrition of the vessel thus detached from the surrounding tissue is maintained by its accompanying nutrient artery, though if the abscess is large there is danger of a deficient supply. The frequent presence of such arteries traversing an abscess should make the surgeon careful how he breaks down the bright pink bands, occasionally seen to stretch across such cavities.

Internal arteritis, or *inflammation of the internal coat of an artery*, is incomparably more serious and mainly because it determines the coagulation of the contained blood and consequent *plugging* of the vessel. This is but one manifestation of the general law that in inflamed tissues the fibrine forming elements and nuclei are produced in excess, and when blood comes in contact with these it tends to coagulate (thrombosis). On the other hand, the inflammation in the arterial coats may ensue from the

pressure of a blood clot formed in the vein or heart and carried on with the current until it reaches an artery too small to admit it (embolism).

The inflammation may be confined to a limited space as when an artery is bruised, stretched so as to tear through its inner coats, or interrupted by a ligature. It may on the other hand be diffused over a greater extent of the vessel, and in some cases, two inflamed portions are separated by intervals of sound artery.

Anatomical features of the inflamed artery. In active inflammation of the internal coats of an artery, it contains blood clots, and if the inflamed surface is not very limited in extent the vessel is completely plugged and the clot forms up to its nearest transverse branch on the cardiac side, precisely as if the artery had been tied. The resulting clot is sometimes tubular, so that an impaired circulation is still carried on. The clot varies in length according to the extent of vessel inflamed, or the distance from the inflamed spot to the nearest diverging branch. The clot is usually fusiform in outline and is firmly attached throughout more or less of its diameter, and occasionally so firmly that it is all but inseparable from the serous membrane. The narrowed ends of the clot mostly float free in the liquid blood, and portions from the end most distant from the heart will sometimes get detached, and by blocking up smaller arteries give rise to new centres of disease. This is a true instance of *embolism* or *plugging*.

The clot has nearly always a grayish or yellowish white color in the larger vessels, such as the posterior aorta, and an uniform pink or red streaked with yellow in the small. It is possessed of great firmness and elasticity. That portion of the surface which was not attached to the arterial walls, during life, is clear, smooth and glistening, while the portion which adhered to these walls is rough, irregular and broken into shreds. It is usually composed of concentric layers, showing its mode of formation.

Clots of this kind analyzed by M. M. Lassaigne and Clement were composed of water 74 parts ; fibrine and albumen 25 parts ; and alkaline salts 1 part.

The vessels filled by these clots are very irregular in their outline, being thick and bulging at one point and thin and constricted at another. The outer coat is rarely the seat of morbid change,

though it sometimes shows branching redness and thickening from exuded lymph. The internal coat where the clot was attached is intensely and unnaturally red, and a rough granular surface has taken the place of the healthy, smooth glistening appearance. In old standing cases the clots can only be separated from such surfaces by dissection with the knife. Other portions of the surface than those to which the clot adheres are usually smooth and polished, though rough granular and injected patches are sometimes met with independently of clots.

The muscles formerly supplied with blood by the obstructed arteries are pale, discolored, unnaturally firm, and if some time has elapsed since the plugging, their striated fibrous structure is made out with difficulty.

Causes. The causes of arteritis are often obscure. Goubaux conceived that it was frequently determined by extreme muscular tension. In support of this view he adduced the facts that it has been mainly observed in the horse, in which such stretching of the muscles is greatest, and that its most common seats have been where the muscles and vessels are most liable to stretching. Thus it is frequent in the posterior aorta towards its termination or in other words where the adjacent muscles (psœæ) are very liable to laceration from slipping backward or from efforts to disengage the limbs when fixed in soft ground; the femoral and axillary arteries are likewise frequent seats of inflammation and are likely to be overstretched when the limbs slip outwards.

Embolism or Plugging of the arteries must be accepted as another cause. This is referred to under *endocarditis*, as an occasional consequence of the detachment of clots and fibrinous substances from the internal membrane of the heart. The detached mass in this case passes from the heart into the aorta and thence through its divisions until it reaches a vessel too small to receive it, when it is at once arrested and determines inflammatory action in the plugged vessel. When arrested in some soft organ, such as the lungs, liver or brain, the resulting inflammation often gives rise to extensive suppuration and abscess. In other situations its effects may be confined to inflammation, the shutting off of blood from particular parts, the impairment or loss of their function and nutrition, and finally atrophy and degeneration.

But the heart is not always the primary source of such clots. Virchow and others have demonstrated, by *post mortem* examinations in cases of plugging, and by a number of experiments on the lower animals, not only that such clots may have their place of nativity in some distant and diseased part of the body and proceed in the veins to the heart and lungs; whence their products pass through the arteries to other distant parts of the body where they plug the vessels and induce a train of morbid changes; but that such embolism, arteritis and abscesses can be produced at will by the introduction into the circulation of solid and insoluble (infecting) bodies. Fragments of decaying and suppurating tissue and the elements of tubercle, cancer etc. may be thus easily carried onward in the current of the circulation, and reproduce themselves at those points where their course is arrested. This mode is one in which secondary deposits of these morbid matters are determined. Embolism and arteritis in the body and limbs, occurring in this way, necessarily have their point of departure in pre-existing disease of the lungs or left heart. The clots, loosened from the capillaries or veins of the lungs, are carried through the left side of the heart into the arteries of the body at large to be arrested in some of the smaller vessels. I have seen plugging of the digital arteries of the hind limbs to occur in this way, in a horse that had been suffering from inflamed lungs.

Microbes and toxins may pass harmlessly through healthy parts, including the pulmonic circulation, to establish colonies and embolism beyond, where the tissues have become debilitated. Thus Gamgee records a case of embolism of the anterior mesenteric, right external iliac and right femoral arteries, supervening on an attack of strangles.

Symptoms of acute arteritis. These consist largely in impaired muscular power in the part which the artery supplied, indications of acute local suffering, such as trembling and tenderness to the touch, if the obstructed vessel lies within reach, it can be felt as an exquisitely tender cord-like mass, and the limb on the distal side of the embolism and dependent on the diseased vessel for its blood supply is anæmic and cold. In the distal portion of the embolic artery and its branches pulsation has ceased. If the lesion is extensive there may be more or less fever, but

a limited arteritis in a small vessel may escape this complication. If the disease is of long standing there is atrophy of the tissues formerly supplied by the embolic vessels. The secondary derangement of nutrition and function are as varied as the organs affected and will be noted below in the special article on thrombosis and embolism.

Chronic arteritis. Atheroma. This is an indolent inflammation supposed to result mainly from strain and overwork, and manifested by thickening and clouding of the serosa, with cell proliferation, softening and fatty degeneration. The diseased substance becomes soft, pultaceous, slightly greasy, and under molecular degeneration it breaks up and is even in part washed on in the blood stream. Other degeneration may occur in the inflamed walls of the artery. The exudate may become organized, constituting fibrous thickening. It may become the seat of calcareous degeneration. It may yield to the blood pressure, becoming slowly attenuated (atrophy), and even dilated (aneurism by dilatation). As a cause of chronic internal arteritis in the horse, should be named the presence, in the vessels, of the larva of the *sclerostoma equinum*. The posterior aorta and anterior mesenteric artery, which are the most commonly infested by these parasites, are frequently attenuated, dilated, thickened or calcified in this connection.

Treatment. Acute arteritis should be treated like any other local inflammation, by rest, soothing applications (fomentations, astringents, icebags), and alkaline salts. It has been proposed to manipulate the affected artery and contained thrombus, but this can only tend to block the smaller arteries farther on, and perhaps with even more injurious results. The liberal use of alkalis, on the other hand, if effective in dissolving any portion of the clot, returns this to the blood stream in a condition that need not endanger further embolism. The agents usually employed are carbonates of ammonia, potash or soda, and iodide of potassium.

INFLAMMATION OF THE AORTA. AORTITIS.

Horse. *Causes.* This has been traced to undue stretching of the vessel, as in slipping of the hind feet backward, or kicking out and very high (Goubaux), sudden falls, sprains of the back (Sabourine), penetrating wounds (Bürmann), irregular strangles with abscess, rheumatism (Liard), endocarditis extending to the vessel (Trasbot), atheroma (Axe), and the larva of sclerostoma equinum or other worm.

Lesions. These are essentially those already described under arteritis. The seat of aortitis is usually a limited section of the vessel, and though in case of sclerostoma it may be very extended, yet as a rule it is confined to the lumbar region, near the points of origin of the anterior and posterior mesenteric arteries. The secondary lesions may be very varied; embolism of one renal artery with atrophy of the corresponding kidney, and enlargement of the other renal vessel and hypertrophy of its kidney; embolism of the artery of the testis or ovary with atrophy of the organ; embolism of the coeliac axis, or of the anterior or posterior mesenteric artery, with violent, perhaps fatal colic; embolism of the external iliac artery or its branches with intermittent paresis of the muscles of that limb occurring under exercise; embolism of the hepatic or renal artery with consequent dropsy; hypertrophy of the left ventricle through the demand for more force to overcome the obstruction in the inflamed artery.

Symptoms are very indefinite, unless plugging of the iliacs and their branches leads to intermittent lameness and even then the aortitis would only be an inference, but by no means proved.

Cattle. *Causes.* Tuberculosis is the most common cause, the intima of the aorta showing small nodular elevations like a small pea, with tubercle bacilli and caseated centre, or ulcerations, or calcifications. The vegetations may extend into vessels originating from the aorta, or may block the openings of such trunks and give rise to various disorders.

Dog. *Causes.* Aortitis has been especially noticed as the result of parasitism and particularly of the spiroptera sanguinolenta or less frequently the filaria immitis. There may be diseases of tricuspid or aortic valves, and vertigo or syncope may result.

Prevention. *Treatment* is out of the question. *Prevention* would be possible in the case of the parasitic cases by keeping the horse's intestines free from the sclerostoma, and by the removal, from kennels and localities, of dogs suffering from the blood strongyle and filaria.

THROMBOSIS AND EMBOLISM.

Definition. Thrombosis: clotting in the vessel. Embolism: plugging of the vessel. Thrombus may form in any bloodvessel. Embolism occurs in arteries. Clot follows the blood current. Causes of clotting: fibrinogen, paraglobulin, fibrine ferment; foreign bodies; parasites; air; blood that has been exposed, (transfusion; aerial germs; disease germs; chemical coagulants; high and low temperatures; breaches of endothelium, congestion or inflammation of the serosa; stasis of blood and extension of clot; ligature near a branch vessel; deoxidation and carbonization of blood, marasmus; neoplasms; traumas of the vascular coats; infarction. Causes of blocking: disintegration of clots, softening, liquefaction, ulceration, action of microbes; excess of white globules; air; fat; parasites. Pathogenesis: complete occlusion of vessel; infarction; sequestrum; collateral circulation; embolism of external iliac or femoral artery; effects on pulse; during rest; atrophy; lameness comes on with exertion, disappears under rest; circulation inadequate to sustain active function. Embolism of internal iliac artery; effect on pulse; on tail and pelvic organs. Embolism of axillary artery; effect on pulse, action, nutrition. Embolism of mesenteric artery; verminous; effects on innervation and circulation; spasms, congestions, paresis; involution. Treatment: expectant; alkalies; gentle exercise, time.

Definition. Thrombosis is the blocking of a blood vessel by a clot formed in its interior by the deposition of layer above layer on its inner coat.

Embolism is the blocking of a bloodvessel by a clot or other solid body formed at a distant point of the circulation, floated on in the blood stream, and arrested when it reaches a vessel too small to transmit it.

A *thrombus* may be formed at any point of the circulatory apparatus (heart, arteries, veins) whenever the conditions are such as to determine coagulation of the blood. An *embolism*, on the contrary, is a disease of the arteries, since in these the blood current, proceeding centrifugally from the greater to the lesser, inevitably carries the moving solid to a point too narrow to allow of

its further progress. Thus clots originating in the systemic veins or right heart pass to the lungs and produce embolism of the pulmonary arteries, whereas those formed in the pulmonary vein or left heart are arrested in some part of the systemic arteries. Clots formed in the portal vein, however, are arrested in the hepatic vessels into which that trunk breaks up.

Causes of Thrombus. The production of a thrombus may be due to the condition of the blood or of the vessels. The researches of Buchanan, Schmidt, Hammersten and others, show that two albuminoid elements, fibrinogen and paraglobulin, present in the living blood, and a fibrine ferment mainly derived from the white corpuscles in process of change or destruction, determine powerfully the formation of fibrine and clot. Hewson, Brücke and Lister have shown that blood may be maintained fluid for many hours in an unimpaired vein, or turtle's heart, though it may have been removed from the body, the important condition being that the vein shall retain its vitality and suffer no derangement of its endothelium. Lister has even shown that blood may remain fluid for many hours in a sterilized glass tube which has been filled by passing the tube carefully into such a vein without disturbing its lining membrane, or imparting motion to the liquid. In such a case a thin film of coagulum only, forms on the interior of the glass tube. In healthy blood, without addition of any extraneous matter, and kept perfectly still, the plasms and globules retain their integrity, and the former its fluidity for a length of time. But if shed into a basin it coagulates at once.

a. *Changes in the blood.* Contact with foreign bodies generally determines this change and prompt coagulation. Transfixing the artery with a needle, even a silver one, the entrance of parasites (actinomyces, strongyli, filaria), the presence of pus, and of certain infecting microbes and their products, the introduction of solid particles and even of air into the vessels, the transfusion of blood which has been exposed so as to receive aerial germs, or which contains microscopic clots, or the globules of which have become disintegrated so as to give up the globulin, or modified by contact with a basin or other vessel, even the transfusion of defibrinated blood may cause coagulation. The danger is always greater if the blood is drawn from a different genus and unfitted

to live in the blood of the recipient. Disease germs are especially dangerous if adapted to colonize the serosa of the vessel and destroy its epithelium. A decrease of the density of blood, favors coagulation, a lowering of one thousandth rendering it syrupy, and various chemical agents induce or favor coagulation, thus acetic acid, valerianic acid, alcohol, the salts of iron, and above all the salts of lime act in this way. Very high and low temperatures throw down the fibrine as a grumous precipitate, but the clot remains soft.

b. *Changes in the vessels* Any disturbance or alteration of the endothelium sets free the so-called fibrine ferment and precipitates coagulation. Thus Lister found that contact of ammonia with the interior of an otherwise living vein caused thrombus. So in all endarteritis and phlebitis, coagulation takes place on the serosa and quickly blocks the vessel. Even in the capillary vessels the same principle holds, and in inflammation minute coagula (thrombi) form in the capillary network throughout the whole inflamed area. This explains not only the capillary blood stasis but the thrombosis of inflamed arteries and veins. In these two latter the clot increases and extends in the direction of blood stasis:—in the artery toward the heart as far as the next collateral branch, and in the veins away from the heart as far as to the next collateral trunk. On the distal side of the arterial thrombus the blood flows off freely toward the capillaries, but on the proximal or cardiac side it is absolutely stagnant up to the next branch through which it can freely flow into the capillary plexus. Into this stagnant blood the fibrine ferment, produced by the altered white globules in the clot already formed, slowly extends until the whole has formed a firm coagulum. Beyond this the actively moving blood carries off and dilutes this ferment so rapidly that it can exert no appreciable effect on the fibrine-forming elements. The principle is an important one in surgery, as the clot formed instead of a ligature will be extensive in proportion to the distance from the first collateral trunk, and in inverse proportion to this clot will be the danger of secondary hemorrhage. In veins the same rule holds, with the difference that as the blood is flowing toward the heart it empties the vessel on the cardiac side, and stagnates on the distal side up to the next collateral branch. Hence it is that a thrombus in a vein

always extends away from the heart, while that of the artery extends toward it.

Another cause of coagulation is the deoxidation of the blood and the excess of carbon dioxide. This occurs in the stagnant blood in the vessels and above all in the capillaries. The normal trophic changes in the serosa, fail to take place in contact with blood in this state, and the resulting changes in the white and endothelial cells set free fibrine ferment and determine coagulation. Stasis of the blood from any cause (ligature, pressure, embolism) tends to this condition and the extension of the coagulum.

A thrombosis of marasmus has been observed in anæmic and debilitating diseases, and apart from the microbial invasions in a certain number of those affections, this may be looked on as due in part to the lessened density and other changes in the blood and to the debility of the serosa of the vessels.

The compression of the vascular walls or their penetration by neoplasms, tumors and ulcers, is another cause of coagulation and thrombus, also a varicose or aneurismal dilatation, with weakening of the vascular walls, or dilatation of the heart with structural changes in the endocardium as stated under that heading, or compression of the smaller vessels and capillaries by an exudate in process of organization, or a similar obliteration under the action of extreme cold. Injury to the serosa of the vessel, by stretching, bruising, laceration or section, determines a thrombus starting from the injured endothelium. In the smaller vessels the thrombus is usually deep red from the entangling of a large quantity of red globules, whilst in the larger arteries the greater part of the globules pass on and the coagulum is largely buff or straw colored.

Again, in obstruction in the smaller arteries, the inactive capillary plexus and the tissues beyond are liable to become gorged with blood with excess of red globules, from the adjacent capillary network, constituting *infarction*, and ending in gangrene. In cases in which this is prevented by the action of intense cold the part may remain pale, as *white infarction*.

Causes of Embolism. As already stated embolism results from a detached portion of a thrombus passing to a smaller vessel and obstructing it. Such detachment is favored by molecular soften-

ing, liquefaction or suppuration in the clot or beneath it, by the destructive action of microbes, or by friction or manipulation of the obstructed vessel. Excess of white globules (leukæmia) favors the formation of minute coagula and embolism. Bubbles of air, globules of fat, or cholesterine crystals block the fine pulmonary capillaries, and the débris from atheromatous patches, ulcers, and tumors opening on the inner wall of the artery, form embolism in various parts. Finally parasites, especially the larval strongyli in solipeds and filaria and spiroptera in dogs, themselves obstruct the vessels and determine coagulation.

Pathogenesis. In the larger arteries (aorta, radical stump of the mesenteric artery) clots (as from sclerostomes) rarely produce dangerous obstruction. In the smaller vessels stenosis is complete and anæmia and gangrene are liable to occur unless the blood supply is partially maintained by anastomotic vessels. When the embolism affects a number of smaller arteries or capillaries in a vascular organ like the lungs, the blood filters in from the adjacent capillaries, in which circulation is still carried on, and this passes through the softened and ruptured capillary walls so that the tissue is charged with globules and constitutes a *black infarction*. In the lung this usually affects one or several lobulettes, forming a pear-shaped mass corresponding to the distribution of the obstructed vessel. The cut surface is black, compact and granular. The lymph thrown out around it forms an organized fibrous sac, and the enclosed sequestrum undergoes a slow necrobiosis, blanching and liquefaction into a pus-like fluid which is removed by absorption. Such results are met with in the parenchymatous organs (lungs, liver, spleen, kidneys, etc.) and less frequently in the limbs. The symptoms will correspond to the particular organ invaded.

In the fore or hind limbs the result is usually less radical. The vessels below the obstructed trunk are connected more or less freely by anastomosing branches, so that the circulation in the tissues below, though somewhat restricted, remains active enough to sustain a fair measure of nutrition. Apart from the suffering, attendant on the preliminary inflammation, the morbid phenomena are largely confined to the absence of pulsation in the lower part of the limb and the inability of the muscles to sustain active contraction.

Chronic Embolism of the External Iliac or Femoral Artery. In this condition the pulsations in the digital arteries are imperceptible, if it has been of long standing there may be obvious atrophy of the muscles of the thigh, but when standing quietly or walking, there is usually no lameness. In continuous rapid walking and, above all, in the trot, however, the patient soon begins to halt on the affected limb, and this rapidly increases, the joints bending under his weight, the toe dragging and the animal threatening to drop altogether. If stopped and allowed to rest for ten or fifteen minutes he gradually recovers and may be led quietly back to his stable without a sign of lameness. But if again trotted fifty or one hundred paces, the lameness develops anew and disappears in the same way when left at rest. The circulation in the muscles is enough for a moderate nutrition but altogether inadequate to sustain active work.

Chronic Embolism of the Internal Iliac Artery. In this case the control of the muscles of the limb may be perfect but there is some indication of paresis of tail, bladder, rectum and anus. Impaction of the rectum is liable to occur. By examination through the rectum the pulsations are felt to be strong in the aorta and external iliac, but imperceptible in the internal iliac blocked by the embolus.

Chronic Embolism of the Axillary Artery. Here there are the same general symptoms, the absence of the radial and digital pulsations, the wasting of the muscles of the forearm, and the intermittent lameness, developed rapidly by exercise and recovering promptly under rest.

Acute Embolism of the Mesenteric Arteries. This will be fully treated under the title of verminous colic in solipeds. The blocking of the branches, usually of the anterior mesenteric artery, leads to derangement of the innervation, congestions, spasms, involutions and other disorders. The presence of the sclerostomes in the fæces, the general symptoms of intestinal worms, and the recurrence of the indigestions and spasms would serve to indicate the nature of the complaint.

Treatment of Chronic Embolism. As affecting the arteries of the limbs, the repair must be largely left to nature, and we must place the patient in a condition, favorable to such repair. Except in the early stages absolute rest is not necessary. Gentle

exercise stimulating to a freer circulation solicits a slow enlargement of the anastomosing vessels (arterial or capillary), and when this has reached a given stage, weak pulsations may again be felt in the vessels beyond and the muscles will once more stand moderate work without lameness. Alkalies and iodide of potassium may be given to solicit solution of the clot, but this can rarely be counted on to the extent of rendering the vessels once more pervious. A small paddock in which the patient can move around quietly is desirable, and in a few months a tolerable recovery may have taken place. Avoid manipulation; detached fragments will cause other embolisms.

Embolism in other organs must be treated on the same general expectant method, and a considerable time is usually necessary to secure a fair recovery.

ANEURISM.

Definition. Divisions : true, false, dissecting, arterio-venous, mixed, traumatic, spontaneous. Causes : violence, rupture, debilitated vascular walls, strains, stretching, force of blood current, overloaded intestine, stranguyly, contiguous inflammation, embolism, microbial invasion of the walls, arteritis, concussion. Symptoms : soft tumor pulsating with the heart, a double rushing sound, diagnosis from abscess, nervous disorders through pressure, cramps, palpitations. Treatment, when desirable : rest, moderate, laxative diet, iodides, bromides, icebags, compression, ligature, galvanopuncture, wire coils, injections.

Definition. A pulsating swelling on an artery, consisting of a sac filled with arterial blood.

Divisions. A true aneurism (aneurism by dilatation, arteriectasis) is a simple dilatation of the artery, the tumor being surrounded on all sides by the distended arterial walls. It is usually fusiform or cylindroid, but may have the form of a more or less rounded sac.

A false aneurism is where the wall of the artery has been broken and the blood is enclosed in an adjacent sac of condensed connective tissue which communicates with the interior of the vessel. The same name has been given to cases in which the inner coat only has given way, and the middle and outer coats constitute the walls of the sac. From its liability to extend and separate the tissues, this is further known as a dissecting aneurism. Arterio-venous aneurism in which an intervening sac communicates with both artery and vein, has been found in the human subject.

Mixed aneurisms are those in which a dilatation of the artery is complicated by the presence of an outside pouch.

A distinction has also been made according to origin into *traumatic* and *spontaneous*. The former is of necessity *false*, whereas the latter may be *false* or *true*.

Causes. Apart from rupture of the arterial coat by direct violence, the common cause is a debility and loss of resistance in the walls. In horses a far larger proportion of aneurisms are deep-seated than in man, in whom forced muscular effort is less common. Yet even in horses the most common seat—the posterior aorta—is liable to overstretching and to inflammation and soften-

ing by reason of contiguity to dorsal sprains. The posterior aorta too, from its size and direction on leaving the heart, is in the direct line of the strongest blood current, and under long continued, forced and violent efforts (as in racing, hunting and heavy uphill draughts), has to sustain an extraordinary blood pressure. Bouley claims as an additional cause the pressure of a loaded colon. This is also the point of all others where the vessels suffer from the presence of the larval sclerostomata. From whatever cause originating, congestion of the arterial coats leads to more or less attenuation, softening or lack of cohesion, and they tend to yield under the blood pressure. Similar conditions operate on the smaller vessels in different parts of the body, and thus overstretching, contiguous inflammation, and excessive blood pressure cause such lesions in the chest, trunk and limbs.

Another cause is embolism which, by blocking an artery, at once increases the tension in the vessel on the cardiac side of the obstruction, and develops inflammation in the arterial coats, robbing them of their cohesion and resisting power.

Eppinger has shown the importance of infecting microbes in weakening the arterial walls and predisposing to aneurism.

The larval sclerostoma equinum already referred to is the most potent factor in solipeds. They accumulate in the anterior mesenteric artery, leading to clotting of the blood, inflammation of the serous coat, and dilatation, so that in some verminous localities nearly every old horse shows a lesion of this vessel.

All forms of arteritis, and disease of the vascular walls which entail attenuation or weakening, predispose to aneurism.

Of direct traumatism may be mentioned an aneurism of the arch of the aorta in a horse struck by a wagon pole, during a sharp descent (Jacob), and two with aortic aneurism after violent blows on the back with shafts of wagons.

Symptoms. An aneurism within reach of the hand is to be recognized primarily by the pulsation of the swelling synchronously with the beats of the heart, and by a double rushing sound with each beat of the heart, observed on auscultation. An abscess over a large artery lying on a bone may pulsate but it is to be distinguished by the presence of a single in place of a double rushing sound on auscultation, by the impossibility of causing more or less complete collapse under pressure, and by the history of

an active phlegmonous inflammation followed by softening which steadily extends from the centre of the previously dense mass. In a case of aneurism of the gluteal artery of the horse reported by King and in one observed by the author, the symptoms were unmistakable. Similar examples on the popliteal artery and others failed to be recognized during life though attended by lameness.

In internal aneurism the symptoms are mostly indefinite. Ollivier found tympany and vomiting in a goat which at the necropsy showed an aneurism of the anterior aorta as large as the closed fist and enclosing a sewing needle. A more careful diagnosis should have detected a retarding of the maxillary pulse and a double rush over the carotid with each beat of the heart. Pressure on the vagus doubtless led to the symptoms noticed. In aneurism of the posterior aorta there have been noticed a loss of life and energy, dullness, lack of appetite and stiffness of the loins, torpor of the bowels, expulsion of fæces with effort and groaning, intermittent colics, lameness in one or both hind limbs, and finally cramps in the hind limbs, and palpitations. In one case Maillet was able to reach the aneurismal tumor through the rectum.

Treatment. The treatment of internal aneurism will be rarely called for in the lower animals, as the disease is not often diagnosed, is usually beyond reach of mechanical applications, and survival without certain power of endurance would seldom be desirable. In some valuable breeding animals it might be worth while to seek prolongation of life. The most promising measures are absolute rest, and low, non-stimulating diet of a laxative nature and in small bulk. Iodide of potassium is often useful in man, and although in the lower animals there is not the excuse of specific disease, yet the rest to the circulation and reduction of blood tension are not to be undervalued. Bromides may be given with the same object.

Other measures applicable only to aneurisms within reach and essentially of a surgical nature include: ice bags and compression. The compression should as a rule begin at the distal end of the limb and be concentrated by suitably shaped pads on the swellings. Ligature of the diseased artery above or below or both above and below the tumor. Galvano-puncture of the aneu-

rism with the object of inducing coagulation. The introduction of coils of fine wire through a hypodermic needle with the same object in view. In both horse and dog the persistent compression with the finger, seconded as it is by the plasticity of the blood has succeeded in checking the flow from large arterial orifices, and offers great encouragement in the application of this measure to aneurisms. The injection into the sac, of tincture of chloride of iron, with firm compression to prevent motion of the blood, is another available resort.

ARTERIO-SCLEROSIS.

Fibrous thickening of the arterial coats and calcification are well known lesions in the posterior aorta, particularly of the horse. Commencing in congestion or degeneration, which lessens the resistance of the vascular walls, the condition tends to dilatation, and if this is checked by compensatory thickening, sclerosis is induced. The combination of a slight fusiform dilatation and fibrous or calcareous sclerosis is well known in the posterior aorta of the horse. Unless it advances to marked aneurismal dilatation, the condition is not often recognized. If diagnosed, rest and quieting of the circulation are especially indicated. Should it occur in other parts of the body the symptoms would correspond to the organ invaded.

ANGEIOMA. CIRCOID ANEURISM. ANEURISM BY
ANASTOMOSIS. VENOUS TUMOR. NEVUS.

These are forms of dilatation and elongation of the network of small arteries, or veins, and even of the intervening capillaries. In man, these constitute the unsightly red patches and swellings that appear on the face and hands. In animals with dark skins and hairy covering they can only be recognized by the swelling, the feeling as of a bag of worms when the hand is passed over it, and by the rushing sound when auscultated. The trouble is usually subcutaneous and is essentially a surgical one. The most promising treatment is by persistent pressure, by electric current supplementing the pressure, by electro-puncture, and by injections of muriate of iron. When the nevus is not too extensive, a double thread drawn by a needle through beneath the tumor at short intervals and cut, and the ends of each thread tied together, so as to completely stop circulation, is most effective. The skin, needle, thread and hands should be made thoroughly antiseptic or aseptic.

PHLEBITIS.

Divisions, traumatic and idiopathic. Causes : punctures, defective blood supply in walls, debile coats, thrombus, infection, overstretching, injury or disease of serosa, irritants in blood, microbic infections. Lesions : exudation, cell growth, breaches in serosa. Adhesive phlebitis, desquamation, granulation, occlusion. Suppurative phlebitis, infection, pyæmia, erysipelas, metritis, ulceration, neoplasms, phlebolites. Symptoms : local, firm, corded, swollen vein, extends ectad, venous congestion, dropsy, gangrene, diagnosis from lymphangitis, fever, venous congestion in vicinity. Treatment : germicide, rest, cold, antiseptics, blisters.

Inflammation of veins as seen in the lower animals has usually been a sequel of bleeding and is hence a purely surgical lesion. Animals, as well as man, however, are subject to idiopathic phlebitis which as affecting the deeper seated veins may be held to be a medical subject.

The *causes* of idiopathic phlebitis are varied. Injury to the walls, like the punctures made in bleeding, if they result in the exposure of a raw, and, above all, an inflamed, surface to the blood, tends to the formation of a thrombus, and of local inflammation. Even the inflammation of the outer coat tends in the same way to thrombosis and phlebitis, and the experiment of Nicasse showed that the dissection of its sheath from a vein, thus robbing it of its vascular and nervous supply, promptly induced coagulation of the blood in the denuded part. The debilitated or devitalized walls evidently give off fibrinogen and fibrine ferment in amount that is incompatible with the maintenance of fluidity. All other forms of direct injury to the veins, leading to disturbance of the endothelium or cell enlargement or exudation in the intima, will operate in the same manner. Sometimes, as in puerperal phlebitis, the inflammation, extending from the adjacent tissue to the walls of the veins, determines thrombosis, and the invasion by pus microbes determines suppuration. Bruises, over-stretching, pressure with over-distension, and the circulation in the blood of irritant matters may lead to changes in the wall, thrombus, and inflammation. Such irritants may be septic or other bacterial products, or they may arise from the colonization of bacteria on or in the venous coats with the same final result.

The *lesions* in the vein are often primarily of the nature of exudation and cell growth in the coat, without at first any change in the serosa or endothelium. Later the changes implicate these, and thrombosis follows with one of various ulterior processes.

In **adhesive phlebitis**, which is most frequent as the result of purely mechanical injury, the endothelium is desquamated and granulations from the denuded surface extend into the clot and finally occlude the vein. A recovery takes place by the organization of this new product and the contraction of the vessel into a simple fibrous cord.

In **suppurative phlebitis**, which occurs especially in connection with infection (erysipelas, metritis), the inflammation, though starting in the same way in the vascular coats, advances rapidly to suppuration, and the intima, lying in contact with the resulting thrombus, may become itself the seat of the suppurating process. Cases of this kind are almost of necessity in the nature of an infection and the danger is greatly enhanced. Small abscesses formed in the vascular coats may burst into the vein and passing on with the blood produce general infection (pyæmia). Even when the pus enters the vein at a point covered by the thrombus, it may escape by the partial loosening of the clot from the serosa, or through the interior of a honey-combed coagulum and thus lead to general infection. This is especially liable to follow in erysipelas and metritis, in which the tendency, as in the solid tissues, is to diffuse suppuration without any investing membrane. There are other forms of bacterial colonization of the vascular walls, of ulceration, and of the extension of morbid growths into or through the venous walls, producing inflammation more or less localized, and leading or not to general infection. The presence of phlebolites in the vein is a conceivable source of phlebitis, though no such case has been so far recorded.

The *symptoms* in localized cases of simple adhesive phlebitis may be purely local. The vein if within reach may be felt like a firm, rounded cord, which extends in a direction away from the heart. If there are no free anastomoses with neighboring veins on the distal side of the thrombus, venous congestion and dropsy of the tissues ensue, and in some cases moist gangrene. When, however, such anastomosis is abundant these peripheral symptoms

may be absent, especially if the affected vein returns blood from a higher level than the heart, and then the symptoms are confined to the vein and its immediate surroundings. From lymphangitis which shows similar hard cords, it is distinguished by the absence of an extended network of diseased vessels, by the lack of a diffuse, doughy swelling, and by the fact that the adjacent lymph glands remain free from inflammation, pain and swelling. In the more extended cases there is fever, which may be a very high type and may merge into pyæmia. In deep-seated cases it may be difficult to identify the disease, but it may be suspected if in the course of erysipelas or metritis there is a sudden increase of fever with pain and swelling, and distension of veins leading into the part.

The *treatment* of idiopathic phlebitis is largely that of the particular infecting disease on which it depends. In simple cases due to trauma, absolute rest and the application of ice and antiseptic solutions, or where these cannot be applied, the use of antiseptics internally, will be indicated. Hyposulphite of soda and sulphide of calcium are especially indicated. From the early days of veterinary medicine, flying blisters of Spanish flies, over the inflamed vein or veins, have proved very successful, and under the lead of Nonat the same was since 1858 adopted with gratifying success in the human subject. Abscesses formed in accessible situations should be promptly opened and treated antiseptically, and swelling of the affected part should be checked by elevated position, or if that is impossible, by a smoothly applied bandage. Rubbing and active movement are dangerous, as tending to detach clots which float off to start new emboli and inflammations in the lungs.

VARICOSE VEINS. DILATED VEINS WITH ALTERED WALLS.

Rare in animals. Angioma. Varix. Superficial. Deep. Causes : obstructed circulation, compression, congestion. Symptoms : enlargement, elongation, tortuosity of veins, stiffness, lameness, complications. Treatment : compression, coagulants, cauterization, ligature.

Varix is not so common in the lower animals as in man, and is generally observed in the superficial veins, so that it comes under the domain of surgery. In the form of angioma, which affects the veins, there is extensive dilatation and elongation, but it involves a large group of connecting and anastomosing veins, whereas varix usually affects but one or a few connecting vessels. In the horse, the most common seat of varix is in the saphena vein, as it passes obliquely over the inner side of the hock. Less frequently it appears on the flank or other superficial part. In cattle, the mammary veins are the most frequent seat. Varices, however, occur also in deep-seated veins and in connection with normal venous plexuses, as in the buccal, palatal, and penial. Anatomically they may be simple fusiform dilatations, as in the saphena ; dilated, elongated and tortuous branching trunks, as in the mammary veins ; or dilated veins with thickened walls and pouch-like dilatations.

Causes. There is usually some obstruction to the circulation through the affected vessel, it may be by pressure by a tumor, or a constrained position, obliteration by a phlebitis and thrombus, extension of inflammation from adjacent organs, increased blood pressure by gravitation, or from diseased heart or lungs. Whether from the extension of contagious inflammation, from external pressure, or from blood tension, the morbid process has much in common ; the circulation and nutrition in the vascular walls are interfered with, degenerations set in (softening, fatty, connective tissue), which predisposes to dilatation under the blood pressure. The pouch-like dilatations of the jugular, consequent on bleeding, are essentially traumatic. The impaired innervation which lessens the resistance of the vascular walls is not to be forgotten. Varix of the saphena is usually an attendant or sequel of tibio-tarsal synovitis, and is the result of combined pres-

sure and congestion. Mammary varices are manifestly connected with the congestion and exudations which affect the udder and environment at the time of parturition, or with a casual mammitis.

The *symptoms* in superficial vessels are visible enlargement, and often elongation and tortuous direction of the vein or veins, with or without tenderness. Deep-seated varicosities may be attended by stiffness of the part and a halting in progression with or without pain on pressure. These cases may recover spontaneously, as the result of adhesive phlebitis, or they may develop phlebolites, suppuration, inflammation, ulceration and hemorrhage.

Treatment. Superficial varices have been treated by compression, cauterization, coagulating injections, and ligature. It is not often that interference is demanded, but in such cases, pressure with elastic bandage having failed, ligature, with antiseptic precautions, is indicated.

PHLEBOLITES. CALCAREOUS BODIES IN THE VEINS.

Nature. Location. Mode of formation. Calcareous plates in two inner coats. Phlebotomy. Altered sanguification. Treatment. Extraction.

Calcareous bodies have been repeatedly found in the veins of man, and several observations of the same kind have been made in the horse. Spooner found them in the abdominal veins, and Simmonds in the jugular. Much difference of opinion has existed as to the mode of formation of these bodies, whether by calcareous deposit in a coagulum or by degeneration of a neoplasm in the vascular wall. Andral held the latter opinion, and Tiedemann and Cruveilhier found the bodies connected to the inner coat of the vein by a fine membrane. Morton's cut of one 'of Simmonds' specimen's (Calculus Concretions) shows a structure in successive layers having their centre at one end, evidently corresponding to a former connection by pedicle. Cornil and Ranvier says "sometimes there are seen in chronic varices, calcareous incrustations in the form of plates, nodules or spheres with concentric layers . . . calcareous infiltration is seen in the form of spheres or phlebolites in the varicose diverticula. An extensive calcareous induration, several centimetres in length, is also sometimes observed, the vein being transformed into a calcareous tube with the ramifications also varicose.

The calcareous plates of the vein are developed in the fibrous and internal portion of the middle coat. At the beginning they consist of granules deposited in the fasciculi of the connective tissue or between them; these soon unite and form transparent plates with granular striæ."

Phlebolites in the jugular suggest a connection with the pouch-like dilatations, and transformations in the vascular walls that have been subjected to phlebotomy. It is probable however that there is usually a morbid condition of sanguification and nutrition which predisposes to their formation. In Simmonds' case the jugular was impervious below the bodies, there was hepatitis and arthritis of the fetlock joint.

When recognized during life these may be extracted, with due antiseptic precautions. If the vein can be dispensed with it may be ligatured above and below, if not, an attempt may be made to

preserve it, extracting through a clean-cut longitudinal incision and securing as perfect coaptation of the edges of the wound as possible.

HÆMORRHAGE.

Arterial, venous and capillary hæmorrhage belong almost exclusively to the domain of surgery. Internal hæmorrhages will be considered in connection with the organs in which they take place.

HÆMOPHILIA.

Definition. Causes: lack of plasticity of the blood, thin walls, blood tension, cardiac erethism, hypertrophy and neurosis. Sex. Heredity through the female. Treatment: depletive, styptic, astringent. Transfusion.

This is a constitutional infirmity, usually hereditary and characterized by the occurrence of profuse and continuous bleeding as the result of otherwise insignificant injuries or even apart from any recognizable lesion. It has been attributed to a slowness in coagulation of the blood, but at the start of a hæmorrhage the blood is rich in corpuscles and coagulates firmly. It has also been ascribed to extreme tenuity of the vascular walls, but this has only been met with in a certain proportion of the cases. Another potent factor is a permanent over-filling of the blood-vessels (Immermann, Delafield, Prudden). The same writers attach importance to cardiac erethism, cardiac hypertrophy, and certain neurotic influences which temporarily increase the habitually congestive diathesis. In man the majority of victims have been males, perhaps because most subject to traumatisms. On the contrary the hereditary transmission is mainly through the female members of the family. The families are very prolific, a condition counterbalanced by the death of the majority of the victims at an early age. Among the lower animals it has been observed in horses consequent on castration (Siedamgrotzky, Kohne, Friedberger and Fröhner), setoning (Kohne, Dieckerhoff), and an ulcer of the leg (Kohne).

Treatment consists in combatting plethora and constipation by saline purgatives. The subject should be carefully protected from injuries. Locally use styptics such as matico, adrenalin,

muriate of iron, tannin, alum, solution of gelatin on pledgets with pressure. Internally calcium chloride, thyroid extract, ergot, lead acetate, iron chloride, tannin, alum, acids. Pure sterilized gelatine solution, intravenously, has given good results. Transfusion is a dernier resort. The bloodplates in the transfused blood arrest bleeding and, as it is not resumed for three or four days, the bleeding surfaces will usually have healed and the temporary danger will be over.

DISEASES OF THE BLOOD.

Obscurity of blood changes. Toxic and globulicidal serums. Agglutination. Corpuscular Thrombi. Red globules, biconcave, embryonic. Source. Escape of immature red globules. White globules, eosinophile, neutrophile, uninuclear, multinuclear, lymphocyte, granular amoeboid, strapnucleated. Conditions of increase. Relation to microbes and their products. Blood plates. Destruction of red globules in the liver. Numbers in animals, in different vessels and conditions.

The blood is the common medium through which all nourishment is conveyed to the tissues, all material to the glands for secretion or transformation, and all effete matter to the various excretories for elimination. It is also the carrier of oxygen for the respiration of the tissues, and the seat of changes, as yet little known, effected largely through the white globules. The activities of the various processes, carried on by the fixed tissue cells and nuclei would suggest, that any disease or derangement of these processes would be at once cognizable in changes shown in the blood. Yet so perfect is the balance of sanguification and elimination on the one hand, and of the remaining vital processes on the other, and so multiform and varied are the products of the vital changes, that it has not hitherto been possible to detect in the blood such changes as would identify the great majority with morbid processes. Some morbid changes are however recognizable and it is important that the significance of these should be known.

The blood is a liquid, consisting of a plasma holding in solution serum albumen, serum globulin, fibrine-forming elements, sugar, urea, salts, and a variety of other soluble organic bodies, and floating a series of semi-solid organized bodies, the red and white globules.

CHANGES IN THE VITAL PROPERTIES OF THE SERUM.

Even normal blood serum is often very injurious to another animal and especially to an animal of a different genus. This inimical action is mainly **toxic** or **globulicidal** (**hæmolytic**).

Toxic Serum is often the result of an admixture of toxins, albumoses or ptomains produced as the result of particular infections and thus belongs to the subject of contagious diseases. In other cases it seems to inhere in given genera and to be effective mainly in animals of a different genus. Hence the great importance, when resorting to transfusion of blood, of seeing that the blood is drawn from the same genus of animal into which it is to be introduced. This toxicity, in certain cases, appears to be connected with the coagulation of blood in the capillaries and arterioles.

Globulicidal Serum is distinct from mere toxicity, its action consisting in the destruction of the red globules by liquefaction (**hæmolysis**). Like toxicity this is usually exerted when the serum of one genus of animal is introduced into the blood of another. It seems to be closely allied to the bactericidal property of serum and like the toxic and coagulative action appears to be associated with the presence of a considerable amount of nuclein (Castelino). The globulicidal action is to a certain extent inhibited by heating to 55° or 60° C., while the toxic property is in the main unaffected. In the action of the serum elements the concurrence of various different constituents is often essential to a given result, toxic, cytolytic, globulicidal, agglutinant, precipitating, trophic, secreting, &c. Thus in abnormal products involved, in immunizing the system or rendering it nonvulnerable to a given invasion, the antitoxin proper is inactive, unless there is established an affinity between it and the tissue cell through one or a series of intermediaries (alexin, complement, sensitizing body, &c.), and between it and the invading microbe through one, or a series of other affinities (opsonin, haptophore, agglutinens, hæmolysins, &c.).

AGGLUTINATING ACTION OF SERUM.

The property of blood serum of given diseases, of causing agglutination of the microbe which causes the disease in question, is a remarkable quality now largely availed of for diagnostic purposes. The *Widal test* for typhoid may be applied by diluting the suspected blood 1:10 in a Thomas's hæmatocytometer and adding to this on a glass slide a few drops of an actively motile culture of the bacillus typhosus, when the microbes promptly cease their movements and precipitate in nonmotile clumps, the liquid becoming clear. It is found that even the dead bacillus, killed by heat, agglutinates and clumps in the same way. This test has been successfully applied in the diagnosis of hog cholera (Gilruth) glanders (McFadyen, Schutz, Moore, Giltner), and other microbial affections.

CORPUSCULAR THROMBI FROM SERUM POISONING.

Flexner has shown that some cases of so-called coagula which occur in the heart in experimental ether and alien serum poisoning, are not true clots, but masses of agglutinated red globules and platelets. Experiment shows that a direct relation exists between extensive blood destruction and agglutination thrombosis, and that such agglutinations in blood-vessels and in vitro are one. In many cases the principles, in animal serums, which cause agglutination or retard it, have much influence on hæmorrhage and the destruction of tissues, as seen in bites of venomous insects, snakes, etc., and knowledge is rapidly advancing along those lines.

THE RED GLOBULES. ERYTHROCYTES.

These have a different origin in successive stages of life. In the very early embryo they are found in groups in the mesoderm. In this early life, in many mammals, all the red globules are nucleated (erythroblasts). At a more advanced stage, but still in the early embryo, they are found in great numbers in the liver and lymph nodes as the second point of origin. In the fully developed embryo they are formed mainly in the liver and spleen. In extra-uterine life they are formed in the red bone marrow.

Form. The embryonic red globule, beside being often nucleated, is not usually disc shaped. The red blood globule of extra-uterine life is usually described as a flattened disc, but Lowenhoeck, Rindfleisch, Weidenrich and Lewis claim that this is a *post mortem* distortion, and that the real form of the live corpuscle, as examined in the fresh or circulating blood, is that of a cup, or a hollow sphere with an opening at one side. The absence of a nucleus from the adult red corpuscle further suggests that the central space and the opening are the result of the loss of the nucleus of the spherical red globule of the embryo. In shed blood the red globules shrink into round disc-shaped bodies concave on each of the flattened sides, and with a tendency to accumulate in rouleaux like a roll of coins.

Size. The red globules vary in size from 6μ to 9μ in diameter, average being about 7μ . Where the smallest (microcytes, microblasts, $2-5\mu$) and large (megacytes, $9-12\mu$) are found in the same specimen, the microcytes, are doubtless largely young cells, though there are also cases associated with loss of blood or pernicious anæmia, which would tend to dilution of the blood and expansion of the globules. Other things being equal, the density of the plasma would contract the globules and the dilution of the plasma would enlarge them.

Hæmoglobin. A reduction of the coloring matter (hæmaglobin), so that the central clear area is largely increased, is a common condition in anæmia.

Distortion of the Red Cells (Poikilocytosis) is not uncommon. It may be mere crenation (indentations) of the margin, or as a more definite change, the cell may be pear-shaped, with one or

more rounded projections at the poles. These projections may break loose, forming very small independent cells. Exceptionally, amœboid movement has been claimed in these projections or in the entire cell. Such degenerations and changes have been noted in anæmia, and in protozoan invasion of the blood (Texas fever, surra, nagana, dourine, mal de Caderas, malaria), and, taken along with other symptoms, they may furnish a valuable corroborative means of diagnosis. Examination should be made with freshly drawn blood—that which has stood twelve hours in a vessel usually shows poikilocytosis.

Number of Red Globules. Counting of the red globules is often of material advantage, although it requires elaborate apparatus, skill, accuracy, and time, which few veterinarians can command. The number of red globules per cubic millimetre of blood, in the different domestic animals, in normal condition, has been set down as follows :

Horse.

Maximum 9.934.000 (Burnett, Moore, Haring, Cady), 8.000.000 (Sussdorf).

Minimum 7.148.000 (Burnett, Moore, Haring, Cady), 6.500.000 (Sussdorf).

Average 7.944.714 (Burnett, Moore, Haring, Cady), 7.212.500 (Sussdorf).

Cow.

Maximum 8.712.500 (Hayem).

Minimum 4.200.000 (Malassez).

Average 6.000.000 (Smith and Kilbourne).

} 7.000.000 in winter and
5.000.000 in late autumn
(Burnett).

Sheep.—9.133.000 (Bethe) 12.090.000 (Cohnstein).

Goat.—9-10.000.000 (Sussdorf), 18.000.000 (Malassez), 19.000.000 (Hayem).

Camel.—10.930.000 (Hayem).

Lama.—13.186.000 (Hayem).

Dog.—4.092.000 to 5.644.000 (Stöltzing), 7.418.000 (Lyon)

Cat.—6.857.000 (Sherrington), 9.900.000 (Hayem).

Pig.—5.441.000 (Stöltzing), 8.668.200 (Wendelstadt and Bleibtreu).

Rabbit.—6.845.000 (Cohnstein and Zantz), 7.107.000 (Löwit).

Guineapig.—3,600,000 (Malassez), 5,859,000 (Hayem).

Hen.—2,400,000 (Hayem), 3,100,000 (Malassez).

Swan.—2,189,000 (Hayem).

Ostrich.—1,620,300 (Hayem).

Variation in Number. Physiological variations in numbers are to be taken into account in making deductions from counts of the red globules. The count is made of the number of globules relative to the other blood constituents and thus a variation of the entire quantity of blood in the body, by the increase or decrease of the serum (water) will make a marked relative decrease or increase of globules in the specimen examined. This does not by any means indicate that there are more or less red globules in the blood as a whole. *Sex.* Sexually mature male animals usually have more erythrocytes by 500,000 to 1,000,000 per c. mm. than the females. Storch even found 476,000 per cmm. more in geldings than in mares, but on the other hand 557,000 less in wethers than in ewes. *Pregnancy* has no constant effect on the red globules (Burnett). *Parturition* is usually attended by a decrease which lasts two to four weeks (Burnett and Traum). *Digestion.* From $\frac{1}{2}$ to 1 hour after a meal, the blood globules decrease in a specimen because of the absorption of nutritive liquids from the alimentary canal and consequent dilution of the blood. After 2 to 4 hours they again increase until after the next meal is taken. Prolonged fasting causes a marked relative increase. After 24 hours fasting a dog shows 400,000 to 500,000 per c.mm. increase. *Emaciation.* Similarly, when emaciated from starvation, the red cells were found to continue relatively above normal.

Profuse secretion of liquid. This decreases the water in the blood and not the globules, which become relatively more abundant in any specimen examined (polycythemia). *Profuse perspiration* from violent exertion, or in disease, has a very marked effect. Still more marked are profuse *diarrhœa* and *dysentery*, and inferentially *polyuria*, *Rinderpest*, *intestinal coccidiosis*, *dropsies*, *hydrothorax*, *ascites*, and *lung plague*.

Repeated small hæmorrhages, rob the system of red globules and reduce the power of reproducing them. With the resulting *anæmia* there is a relative reduction in the number of red cells.

The same applies to *wasting diseases* generally. Advanced

tuberculosis, chronic extensive suppurations, acute streptococcus suppurations, septicæmias, generalized cancer, chronic diseases of the alimentary tract associated with great loss of health and vigor, mean marked anæmia and hypocythemia. A bad case of fistulous withers and poll evil showed but 2.634.000 (Moore, Haring, Cady).

Deadly parasitisms, whether of the lungs (strongylosis), bowels (strongylus, sclerostoma, uncinaria, etc.), or liver (distomatosis), have the same effect.

Aspiration of a large ascitic collection causes, in 24 hours, a large relative encrease of red globules (3.280.000 to 5.160.000) (Limbeck, Stünzünz, Gumprecht).

The following agents, introduced intravenously, encreased secretion of lymph, rendering the blood dense and the red globules relatively more numerous: extract of cancer, pepton, tuberculin, hyoscyamus, pneumococcus, hemialbumoses, nuclein, uric acid (Heidenhain, Lowit, Gärtner, Romer).

Emotions have been found to decrease by dilution the number of red cells in a specimen of blood drawn from the skin. The distension of the capillaries in blushing acts in this way. Also the capillary congestion caused by section of the spinal cord above the origin of the splanchnic nerve (Cohnstein, Zunz). The same followed the distension of the capillaries by a warm bath (Ewing). Conversely a cold bath, constricting the cutaneous capillaries, encreased the relative number of red cells. Cohnstein and Zunz held that it was due to the contracted capillaries holding the globules back.

In *chronic stasis in the vessels* from insufficiency of the cardiac valves, cyanosis, dropsy, and other causes, a marked polycythemia is established (Oertes, Grawitz, Peiper). A moderate encrease under illuminating gas poisoning is referred to stasis (Ewing).

Severe vomiting, as in phosphorus poisoning, may cause encrease of the red cells to nearly double their former number (Taussig, V. Jaksch, Limbeck).

The *new born* have polycythemia in ratio with the cyanosis (Ewing).

Altitude determines polycythemia (from 5.000.000 to 8.000.000 in man and animals) (Viault, Egger, Koppe, Wolff), which

slowly diminishes after two weeks (Campbell, Hoagland, Ewing, Müntz, Niault.)

Ether anæsthesia in man was attended by an increase of 149,000 (DaCosta, Kalteyer, Bennett). *Chloroform* anæsthesia for an hour and a half, by an increase of 2,803,000. With severe hæmorrhage added there was a rise of 2,965,000, and in another case 3,082,000 (Moore, Haring, Cady). The increase under chloroform as compared with ether, is probably due to vaso-motor dilatation so that it may be in part set down to the same cause as in the hot bath, disease of cardiac valves, œdema, etc.

Among diseases that lead to extreme loss of red cells must be named:—Anthrax, Texas fever, protozoan ictero-hæmaturia in sheep, dourine, surra, nagana, mal de Caderas and paludal infections in dogs and horses. In these we have the destructive action of the microbe, bacillus or protozoon, on the red globules direct. From the amount of hæmoglobin passed, hæmoglobinuria should be included in this list, yet the number of red cells (8,421,000), sixteen hours after the attack, was quite up to the normal in the case reported by Moore, Haring and Cady.

From this cursory review of the different agencies affecting the number of the red blood-cells, it will be seen that a comprehensive study of the case, its stage, grade and attendant circumstances, is necessary to a diagnosis from the mere count of the red globules. Yet in certain cases of doubt, and in reference to internal diseases especially, it will come in as a valuable corroborative evidence.

LEUCOCYTES. WHITE BLOOD GLOBULES.

The Leucocytes vary widely in health and much more in disease. The average ratio to the red cells may be set down as 1:357 in man, but in domestic animals Nocard found them 1:800 to 1:1100. The variation in different parts of the vascular system and at different times of the day is striking and suggestive. In the splenic vein 1:60; in the hepatic vein 1:170; in the morning fasting 1:716; half an hour after breakfast 1:347; in boys 1:226; in girls 1:397; in men 1:346; in old men 1:381; in menstruating women 1:247; in pregnant women 1:287 (Stricker).

They may number from 5000 to 1500 per cm. They are larger than the red cells, spherical (amœboid), colorless, reticulated and with one or more refractive nuclei. They show not only amœboid movements, but also what are considered as Brownian movements, the latter sometime after having been drawn and perhaps indicating the approaching death of the cell. Their numbers diminish materially in shed blood. They show nodes at the crossing of the fibres of the reticulum and granules.

Varieties of leucocytes. By staining, the leucocytes can be differentiated into at least five different kinds:

- a. Lymphocytes. 4.6 to 11 μ . Horse.
- b. Large mononuclear leucocytes.
- c. Polynuclear neutrophile leucocytes.
- d. Eosinophile leucocytes.
- e. Mast-cells. Basophile.

1st. *Lymphocytes* of variable size, but approximating to the cells of lymph, are the same or slightly larger than red cells (5 to 11 μ) and when stained in slightly neutralized methylin blue show small violet-shaded nuclei. In the horse these make 24 to 35 per cent. of the leucocytes.

2d. *Large mononuclear leucocytes*, of variable size, from the lymphocyte up, but embracing the largest of the white corpuscles, stain faintly red in Ehrlich's triacid solution of fuchsine and methyle green, with nucleus circular, horseshoe-shaped or elongated. In the horse these form from 3 to 4 per cent. of the leucocytes. In the Guinea pig they often show degeneration vacuoles (Burnett).

3d. *The polynuclear leucocytes* are two or three times as large as the red cells (horse 8 to 16 μ) with nucleus of varied form. The reticulum takes basic stains, many of the granules take neutrophile stains. In the horse, 50 to 69 per cent.

4th. *Eosinophile leucocytes* vary in size from that of the lymphocytes to that of the polynuclear cells. Their granules stain red in acid eosin solutions. In the horse, from 3 to 6 per cent.

5th. *Mast cells* (coarsely granular) vary in size, are mononuclear or polynuclear, and contain granules that are strongly basophile—taking alkaline stains. The nucleus usually stains slightly. In the healthy horse there are from .1 to 2.8 per cent. These are tissue cells and when in excess usually imply disease in the bone marrow.

a *Myelocytes*. Granular cells like eosinophiles, with round nucleus.

b. *Plasma cells* of varying size, nongranular, nucleus small, eccentric, takes Ehrlich's triacid stain—brown, methylene blue—deep blue, deepest in the protoplasm. Found in pneumonia, leukæmia and anæmia.

THE BLOOD PLATES. PLATELETS.

These are the third great division of the formed elements in the blood. They are circular or ovoid, colorless, homogenous, or granular, non-nucleated, taking both acid and alkaline stains, and varying from 1 to 3 μ in diameter. They may be scanty in fresh blood and increase by standing, so that they have been looked upon as the debris of broken down globules and particularly of liberated nuclei. In dry specimens, stained by methylene blue, this origin from the red cells can be apparently demonstrated.

Thrombocytes. Spindlecells. Burnett classes the spindle-shaped cells of the chicken with Bloodplates. These are nearly as long and half as broad as the red cell with large central nucleus, granules and frequently vacuoles. Like platelets they tend to collect in clumps, to stain freely, and when removed from the vessels to degenerate and decay.

Blood dust (granules). These are round or spindle-shaped, .25 — 1 μ in diameter, insoluble in acetic acid, alcohol and ether, and uncolored by osmic acid. They resemble granules escaped from the leucocytes.

METHODS OF MAKING BLOOD COUNTS.

A general analysis of the blood is too elaborate to be introduced here, yet the following statement on making counts may be interesting. Take a Thoma's hæmatocytometer, which consists of a capillary tube graduated to two markings, dividing that end of the tube into two equal halves, then a bulb having a capacity 100 times that of the marked tube, and beyond the bulb a prolongation of the tube extended still further by a rubber tube for aspiration. The blood is allowed to pass by capillary attraction up to the first or second mark on the tube, the blood is then wiped from its tip, the diluting fluid is then drawn in by aspiration until the bulb is filled. It is mixed by shaking and is ready for the counting chamber. The diluting fluid may be : mercuric chloride, 5 grams ; sodium sulphate, 5 grams ; sodium chloride, 1 gram ; distilled water, 200 grams. The counting chamber is made with a thick glass slide, on which is cemented a thin glass plate with its central part cut out. The chamber thus formed must be exactly $\frac{1}{10}$ th of a millimeter deep. The floor of the chamber is ruled in nine square millimetres, and the centre square millimeter is subdivided into 400 small squares, each $\frac{1}{400}$ square millimeter. This centre millimeter alone is used in counting the red globules, while the entire chamber is used in making count of the leucocytes. The red cells counted in 100 of the small squares must be multiplied by four to get the number in the millimeter, then as the chamber is only $\frac{1}{10}$ millimeter deep, it must be multiplied by 10 to get the number in the cubic millimeter, and finally, as the blood was diluted 1:100, it must be multiplied by 100 to get the actual number in a cubic millimeter of blood. Thus the count in 100 squares must be multiplied by 4,000 to give the actual number of red cells. If, on the contrary, the blood was only drawn up to the first marking on the pipette instead of the second, the count must be multiplied by 8,000 instead of 4,000 to get the correct result.

Great care is wanted in manipulation to get the requisite amount of blood pure, in filling and covering the cell to avoid air bubbles or overflow which would invalidate the count, and even in dropping out of the pipette the first two or three drops

which are mainly diluted fluid, so that the blood mixture may be secured.

To get reliable results the blood must be drawn from the cutaneous capillaries. In the domestic animals Moore, Haring and Cady found that an incision on the lower surface of the tail, near its root, proved very satisfactory. The skin is first cleaned and sterilized, care being taken to avoid chilling on the one hand or unduly warm applications on the other.

Leucocytes are counted by a special pipette giving a dilution of one to ten and by using a diluting fluid (3 per cent. acetic acid, tinged with gentian violet) which dissolves the red globules, leaving the stained leucocytes to be counted.

LEUCOCYTES IN DISEASE.

Beside the leucocytosis of physiological origin (digestion, pregnancy, newborn), the condition is especially common *after hæmorrhage*, in *cachectic conditions*, *antemortem*, and in connection with inflammations, especially such as affect large lymph plexuses (cellulitis), or are associated with extensive suppuration. The inflammatory leucocytosis was shown in a marked way in twenty-eight diseased horses in which the blood counts were made at the N. Y. S. Veterinary College by Moore, Haring and Cady. The highest record per cmm. of blood from seven healthy horses was but 6,820 per cmm. Four cases of *fistulous withers* or *poll evil* or *both*, made the following records: 7.549; 10.707; 11.000; 15.232. A case of shoulder abscess furnished 10.793; a suppurating œdema 10.623; and an enlarged (cellutitis) hind limb 14.444; a chronic lymphangitis 7.677; sarcoma of the maxilla after operation 9.925; a suppurating odontoma of the maxilla 20.000; a quittor after operation 11.575; infected kick-wound 14.400; case of tendo-vaginitis 16.266; and epithelioma of the sheath 26.666. It is quite true, that these affections were all appreciable by other and obvious symptoms, but they show that the rule as worked out for man is equally applicable to the soliped, and in some cases in which the lesions were less obvious, the leucocytosis coinciding with other indications might furnish valuable corroborative testimony.

When we come to the experimental cases on small, laboratory animals we find the general application of the principle fully sustained. Rogers and Josue, forty-eight hours after the injection of staphylococcus aureus into rabbits, found great encrease of red cells and eosinophiles, but on the third day these had in the main disappeared and neutrophile myelocytes and giant cells formed the bulk of the blood cells in the now greatly hypertrophied vessels. Virulent diphtheria cultures in Guinea-pigs killed the subjects within forty-eight hours, before leucocytosis set in; but in rabbits, which lived somewhat longer after the injection, leucocytosis became well established before death.

As to the *post hæmorrhagic leucocytosis*, Huhnerfauth found that nine dogs which had lost one-fourth of their weight by acute

hæmorrhage, showed on the same day a hypo-leucocytosis but the white cells had greatly increased the following day (maximum 45,000) and considerable increase persisted for three or four weeks. Lion and Rieder had similar experimental results (with a maximum of 62,000), with only one exception. Considering the important rôle taken by leucocytes in phagocytosis, these suggest a measure of support for the now all but discarded phlebotomy in severe inflammations in strong, vigorous systems.

Eosinophilia is found to be a very marked phenomenon in connection with the anæmia of certain parasitisms, as uncinaria, trichina, bothriocephali, tæniæ, strongyli, sclerostomata, ascarides, etc., so that it has been supposed that the production of the eosinophiles is stimulated by toxins which the parasites have generated. In cachetic conditions and suspected parasitism, therefore, a count of these cells may corroborate the conclusion, or otherwise.

In case of double ringbones with severe lameness and other indications of constitutional bone disease, Moore, Haring and Cady found the eosinophiles in as great excess as in helminthiasis. The presence of worms is not specifically excluded, but if they were, this might introduce another line in which blood counts might prove useful.

PLETHORA. POLYÆMIA.

Definition. Transitory only. Causes: kidney disease, drinking freely, rich feeding, profuse secretion, polycythemia, hyperalbuminosa, excess of fibrine, sugar or fat. Ratio of blood to body. Variations of globules. Symptoms, general, local. Appearance of blood. Prevention. Treatment.

Definition. An excess of blood, of the blood globules, or of the albuminoids.

Formerly accorded an important place in pathology, plethora has been entirely eliminated from some recent works. The actual amount of blood varies greatly at different times, rising after a free consumption of food or drink, and falling during a period of abstinence. A healthy activity of the secretory and excretory organs secures a fair uniform average in the plenitude of the circulatory system. Moreover, large variations are not in themselves rapidly injurious. Worm-Müller and Cohnheim introduced into dogs ten or twelve per cent. of the body weight (fifty or eighty per cent. of their blood) of canine blood without inducing fatal results. More than this was fatal. In non-fatal cases a reduction to the normal standard is speedily secured.

But we cannot count on absolute immunity in all circumstances. Disease of the kidneys, or drinking water to excess, determines a surplus of water and urinary salts (serous plethora, polyæmia aquosa). In cases of rapid gain in condition from rich feeding, and above all after profuse watery secretion (diarrhœa, diuresis, perspiration), the red globules are relatively increased (plethora polycythæmica). After hearty feeding there is a large increase of albumins (plethora hyperalbuminosa). Fibrine-forming elements are apparently in excess during rheumatism, pneumonia and other acute inflammations. Sugar is in excess after a saccharine or farinaceous meal, fat in obese individuals, after consumption of fat, after injuries to the bone marrow, and after severe diseases with much destruction of albumen.

The ratio of blood to the body weight is: in birds, 1:12; in Guinea-pig, 1:19; in rabbit, 1:20; in cat, 1:21; in dog, 1:17; in horse, 1:18; in sheep, 1:24; in pig, 1:26; in ox, 1:29 (Colin). As showing the variation under even different normal conditions of the system Bollinger found the blood but 2.2 per cent. of the

body weight in a fat pig, while it was 15.5 per cent. in a draft horse. Colin found it 2.4 per cent. in the fat ox instead of the usual 3.4 per cent.

The excess of red globules and usually also of albumins is seen as a temporary condition, in lean but vigorous animals put suddenly on an abundant diet, rich in assimilable albuminoids, in working animals, put in confinement to feed, and above all in high conditioned cows after an easy parturition, when the uterine blood has been suddenly thrown on the general circulation and the emunctories have failed to establish a balance. Also in the lymphangitis occurring after a day or two of rest, in a horse that has been hard worked and heavily fed.

It should be borne in mind that the number of red globules varies considerably in the different animals. In the dog it was by weight 148.3 grammes per 1000; in the pig 105.7; in the horse 102.9; in the ox 99.71 and in the sheep 98.2 (Andral), Gavarret, Delafond). By count the horse has about 7,500,000 per cubic millimeter and the dog 5,000,000 to 8,000,000.

Symptoms. Under a sudden dangerous increase of the volume or the organic elements of the blood, there are usually dullness, lassitude, dropping of the head, strong, full, hard pulse, extra force in the heart beats, thirst, elevated temperature, and redness of the visible mucous membranes. At first there is no indication of local disease, but unless relief comes by free secretion some local complication is likely to ensue. This may be epistaxis, congestion or apoplexy of the brain, parturition fever, lymphangitis, or congestion of some internal organ, etc. A drop of blood colors deeply the finger or other object, it clots firmly in three to five minutes, and shows more than usual of a buffy coat.

Treatment. As dangerous plethora is usually a very transient condition the main attention should be given to *prevention*, in keeping the diet low and the emunctories active in high conditioned animals thrown idle; in lowering the diet and securing free secretion, or in giving exercise to highly fed, hard worked horses that have been laid off work; in changing only by slow gradations thin, vigorous animals to a rich diet, etc. When the danger is imminent prompt relief can be secured by the liberal abstraction of blood. Purgatives, diuretics, and restricted diet may be applied to less urgent cases.

HYDROÆMIA. ANÆMIA OLIGÆMIA.

Definition. Causes: bleeding, watery repair, hydroæmia, repair of globules, changes in red globules, in bone marrow. Cause of chronicity: profuse secretions; neoplasms; parasites; chronic exhausting diseases; defective diet or hygiene; diseases of jaws or throat; overwork; toxic substances. Symptoms: palid mucosæ, weakness, perspiration, soft tissues, small pulse, palpitation, anæmic heart—arterial and venous murmurs, depilation, indigestion, costiveness, urine clear, abundant, emaciation. Lesions: blood poor in globules, embryonic, and other abnormal red globules, fatty degeneration, blood clot. Treatment: remove causes, diet hygiene, sunshine;

Definition. Bloodlessness; Deficiency of blood; Lack of red blood globules. The last named is the condition to which the term is habitually applied.

Causes. Anæmia is not so much a disease, as a result of a great many debilitating and exhausting conditions. **Hæmorrhage** the most direct cause of anæmia determines at first an actual lack of blood (oligæmia) and of blood pressure, which may be sufficient to cause fainting and death. In case of survival the amount of blood is rapidly made up by absorption from all available sources of liquid in the economy, but the blood so restored is essentially hydroæmic having an excess of water and a lack of globules and dissolved solids. If, however, the loss has been moderate, the quality may be restored in a few days. Buntzen found that after moderate bleeding the volume is restored in a few hours; after a profuse hæmorrhage in 24 to 48 hours. After bleeding to 1.1 to 4.4 per cent. of the body weight the increase of the red globules may be noticed after 24 hours, and is completed in 7 to 34 days. It is noteworthy that during this repair the bone marrow becomes much redder and more cellular, and that new red cells found in the blood are nucleated (Neumann) and contain less hæmogoblin (Ott). The absence of hæmoglobin is nearly in proportion to the amount of the hæmorrhage (Bizzozero, Salvioli). If the hæmorrhage is slow and continuous, this repair is counterbalanced and the anæmia is much more persistent.

Profuse secretion as of milk (cows, goats, ewes, bitches, on poor feeding), of liquid fæces, urine, or pus, often determine a marked and even dangerous anæmia.

The rapid growth of multiple tumors as of *melanosis* in gray horses has been noticed to cause profound anæmia (Bouley).

Perhaps no cause is more potent than the attacks of **parasites** and especially such as live by sucking the blood. The cases of numerous strongyli of the lungs, stomach, and intestine, the trichocephalus, and allied round worms, the trematodes of the liver, and the cytodites of birds furnish striking examples of the bloodless and debilitated condition which they may produce. In man and animals uncinaria cause anæmia in Egypt, Italy (St. Gothard), America, and elsewhere ; the same with plasmodium, pyroplasma, trypanosoma and bothriocephala in different countries. These are believed to operate through toxins as well.

Chronic exhausting diseases, especially those which affect the digestive organs and mesenteric glands, are prolific causes. So with Bright's disease.

Connected with these are **defects in diet or hygiene**. Starvation, unsuitable, innutritious or indigestible food, too laxative food, damp, dark, draughty or unventilated stables, and irregularity in feeding, watering and work, are all potent factors in inducing anæmia.

Diseases of the masticatory apparatus (broken jaw, diseased teeth), preventing the preparation of food, and pharyngeal troubles interfering with deglutition are other causes. Finally overwork is not to be forgotten.

Toxic anæmia may occur from the ingestion of lead, mercury, or arsenic or from glycerine, pyrogallie acid, hæmoglobin, etc., intravenously (Ponfick).

Symptoms. These may be little marked at the outset in slowly developing cases. Extra pallor of the mucous membranes, fatigue and even breathlessness on slight exertion, a small, weak pulse, with a tendency to become rapid, with violent heart beats, when excited.

At a more advanced stage the mucosæ, especially the buccal, are pale and thin, the muscles are soft, flabby and weak, fatigue and perspiration are easily induced, the feet are advanced more nearly in the median line of the body, and the toes strike on any obstacles, the pulse is weak, small and quick, and the heart easily excited even to palpitation, and with an occasional anæmic murmur with the first heart sound. Arterial and venous murmurs may be present. The hairs are easily detached. Appetite and

digestion fail, there is costiveness, a full secretion of urine of a clear aspect, the subcutaneous fat disappears and the skin feels thin and limp (paper skin in sheep), the hair dry and lusterless, the wool flattened (clapped). The weakness and emaciation go on increasing and dropsies appear in the limbs under the trunk and jaw and in the internal cavities.

Pathology. Lesions. Apart from the causes, the morbid conditions are mainly found in the blood. The watery state of the blood, the lack of red globules (even to but 2,000,000 per cubic millimeter), the absence of albumen (76 per 1000 in place of 83), the loose coagulum with excess of buffy coat, and the excess of serum are characteristic. The presence of large, nucleated (myelogenous) red cells, of spherical bodies smaller than the normal red cells (microcytes), and of irregularly shaped red cells (poikilocytes) is characteristic, the latter especially, of pernicious anæmia. As the disease advances fatty degeneration of heart, liver, kidneys, and other organs are complications and tend to aggravate the disease, by counteracting repair of the globules—thus establishing a vicious circle. All the organs are pale and flaccid, the arteries empty, the veins contain a little blood, forming pale clots. In the cases considered, all the result of another disease, the lack of blood and of the solid and vital elements in that which remains, entails imperfect function in all the vital processes, including sanguification itself, and in this way an anæmia once established tends to perpetuate and aggravate itself.

Treatment. The anæmia above considered being largely symptomatic, or resultant from other diseases, the first consideration as regards both prevention and treatment is to prevent or cure such diseases. Where dietetic or hygienic, a liberal diet, and good hygiene will meet every demand in the early stages. In the warm season an open air life is most important. In case of a drain by over-secretion (milk) this must be judiciously checked. In bitches it will often be needful to wean several of the puppies. A rich and very digestible diet (oats, beans, linseed, oil meal, cotton-seed, milk, gruel), in small compass, and suited to the genus and individual, with iron and bitters, and in the herbivora carminatives, will suit many cases. Muriate of iron, with strychnia or nux vomica; iron sulphate, sodium chloride and nux; or dialysed iron or some other soluble ferruginous salt, with

quinia, gentian, or some other bitter will serve a good purpose. For the dog saccharated carbonate of iron or citrate of iron and ammonia with quinia or strychnia, in pill form is convenient. With poor digestion muriatic acid and pepsin may replace the iron at first. Beef teas may often be given with advantage, even to the herbivora, and injections of defibrinated ox blood night and morning have proved of service. Lecithins may be given by the mouth or nuclein intravenously or subcutem. In extreme anæmia, as from hæmorrhage, transfusion, or its equivalent, must be resorted to. A normal saline solution (0.6 per cent. NaCl), boiled, may be thrown into the peritoneum or subcutaneous connective tissue, or defibrinated blood, may be injected into the peritoneum. Transfusion is the dernier resort.

PERNICIOUS ANÆMIA. IDIOPATHIC ANÆMIA.

Definition. Causes, obscure, faulty diet, hygiene, microbes, glycerine, pyrogallie acid, hæmoglobin, deranged sanguification, parasitisms. Symptoms, of anæmia of obscure origin. Treatment as for anemia, special measures for intestinal fermentations, dietetic.

Definition. Anæmia in horses without any demonstrable cause (chronic disease, parasite, etc., etc.)

Causes. As in the corresponding disease in man the real starting point of pernicious anæmia is obscure. Faults in diet and in general hygiene have been adduced, and while in Berne this appears to be sustained, in Ireland, in the poorest classes, the disease is little known, and in Montreal, it finds its victims largely in a class of well to do artisans (Osler). In the horses it is described on all soils, and on the most varied dietry (Bouley and Reynal). Zschokke and Friedberger and Fröhner in cases occurring enzootically in stables, found a minute bacillus in the patient, which would remove these cases into the list of symptomatic anæmia. The same is true of Texas fever and of Surra, nagana, etc., of horses and mules, in which the trypanosoma destroys the red globules. Laveran and Theiler, working separately in South Africa, on a progressive anæmia, traced it to the *Piroplasma equi*, a near relative of the *Piroplasma bigeminum* of Texas fever, and like it transferred from soliped to soliped by ticks. (See Vol. IV. *Piroplasmosis Equi*). Other forms that are apparently purely idiopathic have been attributed to a failure in the cytogenic processes in the bone marrow especially. In 1904-6 Carre and Vallée showed that the disease is transmissible by transfusion or injection subcutem of blood of a victim, and by feeding water or aliment soiled with its excrement, and further that the germ passed through a Pasteur filter and was ultramicroscopic. This suggests a possible source of our American disease (see below) through horses imported for our rapidly developing territories. Pathological investigation has enabled us to differentiate, according to their respective causes, a number of diseases (distomatosis, lung worms, uncinariasis, internal acariasis, chronic trichinosis, strongyliasis, etc.,) which were formerly classed as anæmias, and it seems altogether probable that the onward progress of medi-

cine will enable us to go farther in the same direction and to allot the remaining unclassified anæmias to their proper etiological places. Some may be unrecognized helminthiasis, others microbial disorders, and still others, disorders in nutrition and sanguification from different causes.

The *symptoms* are those of other forms of anæmia, but being more obscure in origin and therefore less open to corrective treatment, it is more likely to grow up to an extreme development and fatal issue. The weak pulse, irritable heart, debility, unsteady walk with the hind limbs, hurried breathing and sweating under exercise, watery, puffy or sunken eyes, dropsies in limbs and dependent parts of the body, progressive emaciation, and weakness are even more marked than in symptomatic anæmia.

The *treatment* is in the main as for the other form alike in its hygienic and medicinal bearing. In man, recoveries have taken place under arsenic combined with iron. Arseniate of potash and ferrum redactum in pill form, or tincture of chloride of iron, and Fowler's solution in food or water, may be continued for a length of time. Phosphated pepsin, and peroxide of hydrogen have seemed to do well in some instances, and phosphorated oil is another resort. W. Hunter attributes idiopathic anæmia to toxins derived from microbes in the alimentary canal, as Sir Andrew Clark ascribes chlorosis to a similar cause. He prescribes betanaphthol as the least soluble and best antiseptic, in a dose of 5 grains daily in mucilage for man (1 drachm for horse or ox). Hunter further found that a farinaceous diet protected the globules against destruction, while a nitrogenous diet favored this. It may be noted that long ago Delafond attributed anæmia in animals to the extension of the use of leguminous fodders which are rich in nitrogen.

PERNICIOUS ANÆMIA IN SOLIPEDS IN N. AMERICA.

Synonyms. *Infectious anæmia. Malarial fever. Swamp fever. River-bottom disease. Loin distemper. Mountain fever.*

Geographical Distribution. Different observers have reported this disease in *Minnesota* (Beebe, Flocken, Mohler), *N. Dakota* (Van Es), *Manitoba*, *Alberta*, *Saskatchewan* (Rutherford, Torrance, Bell), *Wisconsin* (Clark), *S. Dakota*, *Nebraska*, (Peters), *Kansas* (Kinsley), *Washington* (Nelson), *Nevada* (Mack), *Texas* (Knight, Langley, Parker, Francis, Marsteller).

Causes. The great desideratum in regard to this American Anæmia of solipeds is the lack of evidence as to its essential cause. It is mainly found in a given area, pointing to a definite cause, geologic, telluric, climatic, dietetic, racial, microbial, industrial, or otherwise. The width of this area, extending as it does from the Pacific to the Mississippi and Saskatchewan valleys, and from near the semiarctic Hudson Bay to the burning shores of the Mexican Gulf, is almost necessarily exclusive of any common condition in rocks, waters, exhalations, climate, flora, food, race or mode of utilisation, sufficient to account for the maintenance of such a disorder there, especially when we add its absence from other parts of the Continent, having similar conditions. It prevails alike among hard worked farm horses and in the breeding herd most of which have never worn harness, among the young of one or two year old as among the mature and aged, attacks male and female, breeders and nonbreeders, in pasture or stable, on green or dry food, on surface or artesian well water, on native pastures or seeded meadows, on the swampy sea level of the Gulf of Mexico (Francis) on the one hand and on the dry Ruby Mountains, Nevada, 7,000 feet above the sea (Mack) and on the arid Blue Mountains, Walla Walla, Washington (Nelson) on the other, on the evergreen pastures of the winterless Gulf Coast and on the Saskatchewan River where the winter temperature goes down to 40° below zero F.

Two general features stand out prominently :—1st, *it is a disease of hot weather*, and 2nd, *it shows a strong predilection for rich damp soils*.

On the warm Gulf Coast the affection may appear at any

season and inoculation with the blood of an affected animal proves infecting in the cooler seasons, October to April as well as during the hot months. Yet in chronic cases, relapses show a preference for the warm period, June to September (Francis). In the North the affection is looked for from June to September and protracted cases usually improve after cold weather sets in (Mack). Yet in Walla Walla, Washington, under the soft Pacific breezes, it broke out in March and reappeared year by year thereafter (Nelson).

The effect of damp or waterlogged soil is strongly emphasized in the early and favorite name of *swamp fever* by which the stockman voices his sense of the danger of such lands. Such a consensus of opinion by the great majority of suffering stock owners expresses a fact not to be lightly set aside. That it greatly favors the pathogenic factor is undeniable, but, in the present state of our knowledge, with the arid land cases of Nevada and Walla Walla before us, we can hardly maintain it as the essential factor.

Heat and moisture, so notoriously favorable to this form of Anæmia, may hold their bad etiological preëminence as mere accessories. Not only do they relax the system, and impair innervation and metabolism in the animal, tending to the production of hurtful products of indigestion, and abnormal sanguification, with a greater vulnerability by microbes and their products, but they greatly favor the preservation of such microbes outside of the body, as well as the development of invertebrate bearers which carry pathogenic microbes from victim to victim.

This brings us back to the mooted question of *an organized pathogenic microbe*. This has so far eluded the most careful search by our accomplished microscopists and bacteriologists. Still the indications pointing toward an organized pathogenic factor are almost conclusive. The disease is found in certain circumscribed areas only, into some of which it seems to have been introduced only in recent years and where it has maintained its hold since. The conditions in most such pathogenic areas are such as are calculated to specially favor the preservation, propagation and permanence of pathogenic microbes, and of such invertebrates as are known to carry such microbes from animal to animal. The destruction of blood globules (hæmolysis), the extreme anæmia

and emaciation, the lessened innervation and sanguification, the bleeding from mucous and cutaneous surfaces and interstitially, are familiar results of such parasitism. The very tardiness with which one case often follows another in a herd is suggestive of a blood infesting microbe which does not find a ready means of conveyance from animal to animal and may even require a bloodsucking invertebrate bearer to transfer it from the one to the other. Again the habit of chronic cases to establish a series of remissions and intermissions and subsequent relapses agrees perfectly with the doctrine of a blood parasite which encreases in successive swarms with a corresponding number of relapses or recrudescences.

The transfer of the disease from animal to animal by transfusion of blood from the sick to the well (Mohler, Francis, Mack in America following Carré and Vallée in Europe,) appears to have confirmed the doctrine of a blood-infesting microbe. Francis transferred the disease by inoculation with the blood-serum of a sick horse, also to another horse by injecting 1 cc. of the blood of a horse which had had the disease for one year. He thinks the blood infection may continue for life. Inoculation of the blood on a heifer, sheep, goat, pig and dog failed to produce the disease.

Carré and Vallée in Europe and Mohler and Francis in America passed the blood serum through a Pasteur filter and produced the disease in solipeds by injection of the filtrate. On this basis the hypothetical microbe must be accepted as ultra microscopic. It supports the theory of the identity of the affection in the Old World and the New.

There is no conclusive evidence as to an invertebrate bearer. Horses bitten by flies in the vicinity of an affected animal have escaped injury. Francis and Marsteller "have kept healthy horses with diseased ones all summer and fall, allowed them to eat and drink together, and run together in the same pasture, where there were the same flies, ticks and mosquitoes, yet no disease occurred among the healthy ones."

Symptoms. These vary in successive cases and perhaps in different climates or altitudes. There are pronounced and rapidly advancing anæmia, emaciation and weakness, with loss of nervous and muscular energy, more or less fever, and finally œdemas and capillary hæmorrhages. The mucosæ and white parts of the skin become bloodless and pallid, sometimes yellow

or rusty, the head, ears and lips may droop, and the hind limbs sway or move unsteadily. In *Acute Cases* the *temperature* rises notably, reaching 105° to 107° F. though it is subject to extreme variations, 4° to 5° in a few hours, but only to rise again a few hours later. The *pulse* may vary from 40 to 80 and the heart's action may be tumultuous, palpitating and with firm impact against the ribs. There is *venous pulsation* in the lower part of the jugulars. *Respirations* vary from 15 to 40 and are usually labored. Some represent the *appetite* as ravenous but this appears to be especially characteristic of subacute and chronic cases, while in the acute and in a recrudescence of the chronic, food may be entirely refused. During a remission, however, appetite is usually good or craving. Control of the limbs is largely lost, the animal *swaying* or *stagging*, and *emaciation* is *rapid* especially in the hind parts. *Blood* may ooze from pituita and drop from the *nose* or it may escape from the *anus*. There may appear petechiæ on the pituita, or on white parts of the skin and interstitial hæmorrhages or bloody sweat may be shown. *Colicky pains* may be present to the acute stage, and paralytic incontinence or *frequent* or *excessive urination* may be a marked feature. Intermittent—diarrhœa is also an occasional occurrence.

The most striking and conclusive change is in the blood. In acute and rapidly fatal cases this is less marked, but in the subacute and chronic, the difference is obvious. The red globules decrease to one-half (from 8.248.000 to 4.200.000 per c.mm. or even to 2.000.000, and in one case 980.000 per c.mm.) (Mack). Lymphocytes may be increased, polynuclears decreased.

Acute Cases die in one day to three weeks, death being preceded by emaciation, pallid sunken eyes and advancing paralysis, the animal going down and struggling ineffectually to get up. Bleeding from nose and anus is not uncommon.

The *Subacute* and *Chronic Cases* may last for months (over a year even), the initial fever giving place to a remission, the temperature descending to near the normal, the appetite returning, the mucosæ taking on a more rosy hue, the red globules in the blood encreasing, the sunken eyes becoming fuller and clearer, and under good feeding the general condition may improve. But a recrudescence follows, a new access of fever sets in, and all the evil symptoms reappear, leaving the patient worse

than before. It is after a succession of such relapses that the patient shows the extreme reduction of red globules, the most pallid mucosæ, the most marked sinking of the eyes, the œdemas, and the greatest emaciation and weakness (paraplegia). All or nearly all die sooner or later, some estimating the mortality at 90 per cent, while others are sceptical of any really permanent recoveries. Convalescents under exertion quickly show dyspnoea, rapid pulse, paresis, especial posterior, and early exhaustion.

Lesions. On necropsy are found the general lesions of anæmia, thin watery blood, pale muscles, dropsies under the skin, minute hæmorrhages between the muscles, and on the peritoneal aspect of the intestines, the latter of a bright red, engorged spleen and liver, with pectechiæ, lungs with small solidified areas and together with the pericardium punctate blood extravasations, blood-stained pleural and pericardial fluid, petechiæ and massive extravation in right ventricular endocardium, blood-stained bone marrow—notably in the epiphyses of the long bones (femur, humerus), and enlargement and blood-staining of lymphatic glands. The parenchymatous organs, (liver, kidneys, heart), show extensive degeneration, necrotic and, in exceptional cases, amyloid (Mack). Wounds often heal tardily (Mack).

Prevention. Treatment has been attempted mainly with quinia and arsenic, strychnia, glonoin, ol. terebinth, creolin, etc., and more or less credit has been claimed, probably in connection with the remissions in protracted cases.

Intelligent and effective prophylactic measures must be based on a fuller knowledge of the microbian cause, its life out of the equine body, and the mode by which it gains entrance to the victim (food, water, invertebrate bearer). Its indefinite survival in the animal attacked does not encourage the hope of rendering the animal invulnerable by educing its own native powers of defence. For the Nevada cases Mack inclines to the opinion of Carré, Valleé and Ostertag for the European ones, that radical measures will be required to eradicate the disease.

CHRONIC ANÆMIA. DROPSY IN CATTLE AND SHEEP.

Definition. Causes, parasitic and microbial. Symptoms. Treatment.

Definition. A progressive anæmia in ruminants and other animals, resulting in general anasarca, and dropsies of the internal cavities.

In veterinary works published on the European Continent this affection is given a special place apart from the same train of symptoms which mark distomatosis, taeniasis, and strongyliasis. The disease is described as prevailing in wet years, after inundations, when the vegetation is rank and aqueous, and of course largely aquatic, in animals that are turned out in early morning before the dew has evaporated, in the conditions, in other words, that favor the ingestion of parasites. It prevails also in work oxen, fed on the refuse of sugar factories (beets, turnips) in which the nitrogenous materials are held to be deficient, but in Great Britain where cattle are often fattened on an exclusive diet of turnips, containing even a larger proportion of water, this non-parasitic disease is unknown. It is also ascribed to close, ill-ventilated, unwholesome buildings, and to over-kept and tainted fodder, and so far as a separate disease exists, it seems more reasonable to charge it to the toxins produced by bacterial ferments or cryptogams than to causes which elsewhere appear to be inoperative.

The *symptoms* are essentially those of distomatosis, and the *treatment*, apart from the parasitocides, is the same. When helminthiasis can be certainly excluded, *prevention* would include the avoidance of the factory refuse, especially when in a state of decay.

MELANÆMIA. BLACK PIGMENT IN BLOOD.

Definition. Melanin, in normal tissues, abnormal. Melanosis. Bisulphide of carbon subcutem. Decomposition of hæmoglobin in leucocytes. Coloration of tissue.

Definition. Accumulation of granules and scales of blood pigment (melanin) in the circulating fluid, and in various organs (spleen, liver, bone marrow, brain, etc.)

Melanin— $C_{44.2}$, H_3 , $N_{9.9}$, $O_{42.6}$ —or black pigment (a close relative of hæmatin) occurs physiologically in epithelium (choroid, retina, iris, in the deeper layers of epidermis, and on the surface of the dog's lung and of the sheep's brain) and in connective tissue corpuscles (lamina fusca of the choroid).

Pathologically it is found in the blood of the victims of malarious fever, often in great abundance, and in the spleen, liver, bone marrow, brain, lymph glands and some other organs. It is formed abundantly in the black pigment tumors (melanosis) of man and animals, and in extensive melanosis is present in the blood of both man and horse (Schimmeln). So far it has not been found in connection with the extensive destruction of red globules which takes place in anæmia. Schwalba has developed melanæmia experimentally by the hypodermic injection of bisulphide of carbon in rabbits.

According to one view, the melanin is produced in connection with the destruction of red globules in the liver, spleen, etc., and is thence carried into the blood. This is in keeping with the local formation of the pigment in melanosis. Arnstein, however, urges that in malarious cases the destruction of the red cells takes place in the blood, and that the hæmoglobin, absorbed into the leucocytes, is transformed into melanin, and finally deposited in the tissues by the migrating white corpuscle. Why the hæmoglobin set free in anæmia is not similarly transformed, does not appear. The pigmented organ may be quite black in the immediate vicinity of the blood vessels, and in its general aspect in chronic cases, reddish brown, dark gray, or dark olive.

LEUKÆMIA. LEUCOCYTHÆMIA.

Definition. Nature. Result of other morbid processes. Leucocytes, polynuclear. Lymphatic leukæmia. Spleno-myelogenous leukæmia. Leucocytes in each. Loss of amœboid movement. Charcot's crystals, Hæmatoblasts. Cell increase in bone marrow. Myelocytes. Enlarged spleen and lymph glands. Hæmorrhages. Lymphoid growths. Susceptible genera. Causes obscure. Symptoms: palor, listlessness, weakness, apncea, sweating, thirst, emaciation, weak circulation, anæmic murmur, enlarged spleen, bleedings, diarrhœa, dropsy, excess of white globules, reduction of red globules, buffy coat, beaten fibrine is granular, china-white mucosæ, hurried breathing, stertor, deranged digestion, marasmus. Duration. Not inoculable. Treatment, not hopeful, as for anæmia, good hygiene, tonics, stimulants, antiseptics.

Definition. An excessive and persistent increase of the white blood globules, and associated with enlargement of the spleen, liver, lymph glands or bone marrow.

Nature. This must be distinguished from the leucocytosis which occurs during digestion, or that which attends on tuberculosis, glanders, pneumonia, and other extensive inflammations, and profuse suppuration. These forms are transient and the cells are of the polynuclear variety. The cells of leukæmia are various in character, but bear some relation to the particular organ which is the seat of hypertrophy or morbid process.

"In **lymphatic leukæmia** the increase in the number of leucocytes is due to the mononuclear lymphocytes especially of the small form. As many as ninety-five per cent. of the colorless cells may be of this form. In **spleno-myelogenous leukæmia** the eosinophile cells may be especially increased in number, and there are also large leucocytes coming apparently from the marrow of the bones, and called myelocytes. These most nearly resemble the larger lymphocytes of normal blood, but they are usually larger. They have a single large nucleus which stains feebly and their bodies may show neutrophile granules. Larger and smaller nucleated red blood cells may be found in spleno-myelogenous leukæmia. The leucocytes are frequently in a condition of fatty degeneration, and there may be a decrease in the number of red blood cells." (Delafield and Prudden). In splenic leucocythæmia, blood plates may be absent and in lymphatic leucocythæmia, they may be in excess.

Cafavy claims that many of the leucocytes have lost their active amoeboid movements.

Bright white crystals in the form of elongated octahedra are found not only in the blood but in the diseased glands, spleen or marrow (Charcot's crystals). Clusters of discoid hæmatoblasts (blood plates) are present in the blood in variable numbers (Schultze's granule masses).

The bone marrow is marked by an accumulation of spheroidal cells, which tend to pass into a condition of fatty degeneration. Most of them are colorless, larger than the lymphocytes of normal blood and have one large, often vesicular nucleus, staining less highly than the lymphocyte nuclei, and with neutrophile granules in the protoplasm (*myelocytes*). There are besides, nucleated red blood cells, spheroidal cells, containing red blood cells, and Charcot's crystals. The marrow may be uniformly red, mottled gray and red, gray, grayish yellow, or puriform (Delafield and Prudden). This may affect one or many bones. The affected spleen is usually much enlarged, at first uniformly, later unevenly, firm or softened, and with thickened white capsule. The cut surface is smooth brownish red, or yellow, with white lines (thickened trabeculæ) and indistinct Malpighian corpuscles. It contains glutin, glycocoll, hypoxanthin, zanthin, leucin, tyrosin, and lactic, acetic, or formic acids.

The affected lymph glands are somewhat enlarged, red or gray, exceptionally softened or caseated, and otherwise contain an excess of leucocytes.

Slight hæmorrhages may appear in any of these structures. Lymphoid growths may appear in a number of other organs as the liver, heart, lungs, kidneys, bowels, tonsils, the different blood glands, the serôsæ and the retina.

Genera affected. It has been seen mainly in dogs, but also in horse, ox, pig, cat and mouse. Nocard has collected the following cases: horse 9, cattle 6, pig 5, dog 22, cat 1.

Leisering found a horse's spleen weighing 28 kilogrammes. Johne found a pig's spleen of 2.4 kilogrammes.

Causes. The primary causes of leukæmia are unknown. Löwit's protozoon is found also in healthy blood, and the disease is not inoculable. The enlarged liver has resumed its embryonic function of developing nucleated red cells, and contains mitotic

leucocytes and giant cells. The hæmoglobin in the blood is diminished, and the specific gravity of the liquid lowered. The blood is less alkaline, and in some cases it shows less potassium and chlorine, and more phosphorus, sulphur and sodium than normal (Freund and Obermeyer). As in anæmia all unhygienic conditions are invoked as causes. That it is not due to simple hypertrophy or irritation of the leukogenic centers is plain, as it does not follow on ordinary diseases and injuries of these parts, but what is the precise nature of the morbid cause has so far eluded us.

Symptoms. Pallor of the visible mucous membranes, listlessness, lack of energy and endurance, breathlessness and perspiration on the slightest exertion, ardent thirst, rapidly advancing emaciation, unsteady gait, stiffness or lameness, lies most of the time, walks with pendent head, and jaws open, small, weak pulse, anæmic murmur in the heart, enlarged lymph glands, or spleen felt beneath the left lumbar transverse processes in the ox, or in the left hypochondrium in the horse. Bleeding from the nose or elsewhere, slight hæmorrhage into the conjunctiva, irritable conditions of the bowels, diarrhœa and dropsies are suggestive. The blood when obtained in epistaxis or drawn by a needle prick may be pale rose, brownish or grayish brown instead of red, and under the microscope shows the enormous excess of leucocytes—the ratio to the red being sometimes 1:2, or even more, in the human subject. In the domestic animals the following ratios have been made by actual count: 1:85 (Leblanc and Nocard), 1:50, 1:45 (Mauri), 1:20 (Nocard), 1:15 (Siedamgrotzky), 1:12 (Forestier and Laforque). The normal average for the domestic animal according to Nocard is 1:900. This great relative excess of white globules serves to distinguish this malady from anæmia, and its persistency is a means of diagnosis from transient leucocytosis.

The red globules are always reduced in number, in the horse and dog to 5,082,000, and sometimes even to 2,050,000 per cubic millimeter, while the normal is 7,500,000 (Nocard).

In clotting, the blood forms an extensive buffy coat, and in solipeds which normally show this, the blood set in a test tube forms three strata, the upper slightly yellow, semi-transparent and formed of fibrine, a median of a dull, opaque white color and formed mainly of leucocytes and blood plates, and a lower of a violet red and formed mainly of red globules.

The amount of fibrine is variable. It becomes granular when beaten. Albumen is variable but usually reduced.

The visible mucous membranes are bloodless and of a clear porcelain white. The walk becomes weaker, fore feet wide apart and the hind limbs partly flexed, head and neck extended, and breathing labored. The breathing may be with constant stertor, the bowels torpid and tympanitic, or loose and foetid, dropsies and hæmorrhages ensue, and the patient dies in complete marasmus.

Diagnosis is mainly based on the changes in the blood. The leucocytes should reach at least 150,000 per c.mm., and may reach 1,000,000, mostly lymphocytes, though myelocytes (neutrophile) may be 20 to 60 per cent. If there are less than 150,000 leucocytes, and less than 90 per cent. lymphocytes it may be a mere inflammatory leucocytosis. Eosinophile cells may reach 3,000 or even 100,000 in acute cases. Many of them being mononuclear and containing large, deeply staining granules.

Duration. The disease may prove fatal in less than a month, or it may last for three, six or eight months. It is mostly fatal.

Not inoculable. Many attempts have been made to transmit it by inoculation, but in no case with success.

Treatment is not successful. All hygienic measures should be adopted, as for anæmia; open air and sunshine, with protection against chills; the treatment of all complications; iron, bitters, phosphorus, arsenic in particular, electricity to the spleen, massage; oxygen inhalation; and locally, or generally iodide of potassium or mercury compounds.

LYMPHADENOMA. HODGKIN'S DISEASE.

Definition. Relation to leukaemia. Causes. Mainly accessory, State of lymph glands, spleen, liver, bone marrow, intestine, tonsils, thymus, kidneys, liver, lungs, bronchial mucosa, pleura, pericardium, nervous system. Symptoms, as in leukaemia, with adenoid hyperplasia, but little leucocythemia. Relation to glanders. Uric acid, low density, no hippuric acid. Tuberculin and mallein tests. Treatment as in leukaemia. Excisions in cases not constitutional. Phosphorus, phosphide of zinc.

Definition. Hypertrophy of the lymphatic glands with little or no leucocytosis. There may further be lymphoid growths in the liver, spleen, bone marrow and other organs.

The visceral lesions in lymphadenoma do not differ in character from those of leukaemia, and as it does often apparently merge into that disease by the characteristic changes in the blood, it is denied by many that it constitutes a separate pathological entity. In his admirable monograph on leucæmia in the lower animals, Nocard affirms their identity. The main excuse for keeping up an alleged distinction, is the frequent absence of leucocytosis, and this often supervenes after the lymphadenoma has existed for some time.

Causes. As in leucæmia, no definite cause can be found in the majority of cases. An accessory cause can sometimes be observed where a local irritation gives rise to swelling of the adjacent lymphatic glands and this goes on to distinct lymphadenoma.

Lesions in the Lymph Glands. The hyperplasia may affect but a single group of glands, more commonly a number of groups, and often nearly all. In one case only of leukaemia in the lower animals, a dog, has Nocard failed to find the lymph glands affected. In the horse he has found the sublumbar glands alone weighing 14.5 kilogrammes, 11 k. and 8 k. They compressed the posterior aorta and vena cava and had caused extensive ascites.

The enlarged glands are white, gray, or in case of rapid growth, veined or pointed with red; they may be soft or firm according as the hyperplasia has operated most on the trabeculæ or the cells; they are homogeneous throughout. The scraping of the cut surface gives a more or less thick milky juice containing a great number of nucleated or double nucleated lymphocytes, free nuclei and granules which stain strongly.

Hardened sections show an enormous development of the follicles at the expense of the medullary walls, and double nucleated white globules packed in a rich reticulum of adenoid tissue, whilst the blood-vessels in the connective tissue are crowded with white cells, and there are slight ruptures, old or recent.

Lesions of the Spleen. These are nearly always present. Leisering found a horse's spleen over three feet long and 28 lbs. weight, and Nocard one of 13 lbs. Bollinger found a pig's spleen 3½ lbs. Siedamgrotzky found a dog's spleen over 2 lbs. The consistency is usually firm (sometimes soft in dog). Capsule thickened and white, cut surface dry, reddish brown, granular. Malpighian bodies enlarged like a pea, hazel nut or walnut, with contents as in the lymph follicles. The capillaries are enlarged and crowded with white cells.

Lesions in the Liver. The liver is enlarged in one-half of the cases of leukæmia in the lower animals. It has been found to weigh 20 lbs. in the horse, and 4 lbs. in the dog. It is of a grayish brown, or yellowish brown hue, or light red spotted with yellow, or mapped out by anastomosing grayish white lines. There may be enlargement of the acini, or the formation of little nodes of adenoid tissue, or most commonly in the lower animals, there is an adenoid thickening of the bands of connective tissue, extending in from the capsule. These are filled with white cells which stain deeply with carmine. There may also be slight extravasations of blood and infarcts.

Lesions in the Bone Marrow. These noted in the pig by Fürstenberg, and in dogs by Siedamgrotzky, consist in increased vascularity, great cell hyperplasia, and formation of adenoid tissue as described under leukæmia.

Lesions of the Intestine. These commence in the agminated or solitary glands, which become enlarged, causing thickening of the mucous membrane, and later grow out into more or less rounded masses of lymph—adenoid tissue up to an inch in thickness. They are quite subject to ulcerations.

Lesions of the Tonsils. Bollinger, Nocard and Siedamgrotzky found these enlarged in dogs in connection with adenoma of the spleen. They were soft, friable, grayish, and consisted of a very delicate and fragile adenoid tissue.

In one case Siedamgrotzky found adenoid hypertrophy of the

thymus in a cow, and adenoma of the kidneys similar to that of the liver has been noticed.

Similar adenoid hyperplasia has been found in the lungs, the bronchial mucous membrane, the pleura, the mediastinal and bronchial glands, and the pericardium. In man this has invaded the nerve centres, and it seems that at any point where there is a lymph gland or a lymph plexus this adenoid hyperplasia may localize itself.

Symptoms. The general symptoms of failing health are as described in leukæmia. The particular symptoms of this disease consist in the recognition of the adenoid hyperplasia in the absence of a marked leucocytosis. The submaxillary glands are usually the first attacked, and the disease may, in the horse, be confounded with glanders. There is, however, no pituitary discharge nor ulcer, the glands are enlarged symmetrically on the two sides, and a careful search will usually discover other groups with similar symmetrical enlargement. The parotidean, the pharyngeal, the prepectoral, the prescapular, the axillary, the popliteal, the prefemoral, the post and premammary, and the inguinal should be critically examined. The enlarged mesenteric glands may be reached and detected by the hand engaged in the rectum, or in the small animals by external palpation, as may also the enlarged spleen or liver.

The adenoid hyperplasia in the chest offers very obscure and uncertain symptoms. The enlarged bronchial and mediastinal glands may seriously interfere with the functions of the vagus nerve, causing, in cattle, disturbed digestion and rumination and tympanies, in horses stertorous breathing, and in the carnivora and omnivora a tendency to vomiting. In animals generally the pressure on the cardiac nerves leads to great irritability of the heart, and violent action under any exertion. The prominent dyspnœa in the advanced stages may be explained by these thoracic hyperplasias.

Nocard claims that the urine furnishes most important indications in its low specific gravity (horse 1010), its constant acidity, and in the almost entire absence in that of the horse of hippuric acid. When there is any suspicion of tuberculosis or glanders, the tuberculin or mallein test will decide.

Treatment is essentially the same as in leukæmia, and equally

unsatisfactory. Arsenic has in the main given the best results. In the very earliest stages when the granular hyperplasia is confined to one group, excision is advisable. This should be avoided in all cases in which the constitutional symptoms have developed. Phosphorus and phosphide of zinc have seemed beneficial in certain hands. Injections into the glands have so far proved useless. Cod liver oil, internally with phosphorus and locally as an inunction, has appeared useful. Careful diet and genial out-door life are important.

ACUTE LYMPHANGITIS OF PLETHORA IN HORSE. ANGEIOLEUCITIS.

Definition. Symptoms and causes. Genera affected. Causes of plethora : high feeding, work followed by rest. Fever, hurried breathing, strong, rapid pulse, anorexia, stiffness and swelling in a hind limb, inguinal glands, connective tissue engorgement, corded lymphatics, suppuration rare. Mild forms. Lesions, in lymphatic vessels and glands. Chronic cases. Nature, plethoric, lymph excess, stasis, excess of cells and fibrine, immunity of the fore limb. Season of prevalence. Climate. Diagnosis, from farcy, erysipelas, etc. Treatment : exercise, friction, resolvents, purgative, bleeding, diuretics, astringents, iodide, pressure, diet. Prevention. Treatment of chronic cases.

Definition. Inflammation of the lymphatic vessels and glands of one limb usually in connection with rest and high feeding.

Symptoms. This affection is common in heavy draft horses of a lymphatic temperament and kept on high feeding and at hard work. It rarely develops, however, while the subject is kept at steady work. But if, in the midst of such work, the horse is left at rest in the stall over one, two or more days on the same generous diet, he may be found shivering violently, with rapid, labored breathing, high pulse and elevated temperature, symptoms which have been frequently mistaken for those of pneumonia. There is complete anorexia, and often ardent thirst. The patient is indisposed to move and if forced to, it shows lameness in one hind limb with an extraordinary abduction of the member at each step, sometimes so severe as to prevent his putting his full weight upon it. If an examination is now made high up in the groin close outside the inguinal ring, the lymphatic glands will be found to be swollen, hot and tender, so that under even moderate pressure the leg will be lifted and abducted until the patient threatens to fall on the other side.

A little later the shivering may have given way to the hot stage, with it may be general perspiration, and the swelling may have extended down the course of the saphena vein and lymphatics as a distinct ridge, and the lower part of the limb from the foot to the hock may be filled, dropsical and hot. Unless checked the swelling goes on increasing till the lower part of the limb is two or three times its natural thickness, and the swelling has extended well up on the thigh. The swelling has a soft cedema-

tous feeling, easily receiving and retaining the imprint of the finger and is not only hot, but excessively tender. From the margin of the swelling, firm, tender, rounded cords are found to emerge passing upward along the line of the saphena vein and its branches toward the inguinal glands. These represent the swollen and gorged lymphatic trunks, and may often be traced for some distance into the substance of the general engorgement.

When the inflammation is violent, suppuration may ensue at one or several centers, but more commonly the engorgement goes on increasing and when the febrile attack has subsided the limb is left permanently enlarged and correspondingly liable to a second attack.

Milder cases are met with which are perhaps even more misleading. There may be little or no rise of temperature, loss of appetite or general constitutional disturbance, but under some change of regimen and particularly after one or two days of rest the subject becomes lame in one hind limb, without any of the usual injuries to account for it. Examination of the groin shows swelling and tenderness of the external inguinal glands, with or without a tender, cord-like swelling running down from them.

Between these two types may be found all grades of lymphatic inflammation with a varied degree of attendant constitutional disturbance.

Lesions. The coats of the inflamed lymphatic vessels are thickened by exudate and the outer coat is the seat of ramified redness with minute spots of blood extravasation. The inner coat is dull, opaque, or even thickened. The vessel is dilated and its walls friable. The contained lymph in the intervals between the valves has coagulated into a very thin, diffuent, jelly-like clot, which in old standing cases may have become granular. The connective tissue from which these vessels lead is infiltrated with liquid and the lacunar spaces distended. Red patches from blood extravasations are numerous. The external inguinal glands and often the internal and sublumbar are swollen, congested and the seat of active cell hyperplasia. Abscesses are exceptionally seen.

In chronic cases the lymphatic vessels of the affected limb and especially of the lower part which is permanently swollen, are enormously increased in calibre (lymphangiectasis), and have

their walls correspondingly thickened. The connective tissue is the seat of extensive fibrous hyperplasia, and its interstices are greatly enlarged.

Causes. Nature. This disease has not been sufficiently studied to ascertain what toxic agents are produced in the plethoric condition, under the torpid processes of nutrition and sanguification entailed by absolute compulsory rest. A consideration, however, of the relations of the lymph and lymph vessels and glands to other parts will in part explain the pathology of the malady. The lymphatics take their origin in the nuclear spaces of the various tissues, the anastomosing canals of such pericellular spaces, together with the latter forming the actual radicles of this set of vessels. They receive, therefore, the surplus plasma which is not used up by the tissue-cells in performing their trophic, secretory and other functions. This lymph carried on by the *vis a tergo*, muscular compression and other movements, is delayed in the adenoid tissue known as lymph nodes, and especially in the lymph glands, in which the proliferation of lymph cells is mainly carried on. Thus the lymph cells are very scarce in the lymph radicles of the connective and other tissues, and are found in greater numbers after passing through the lymph nodes, and in still greater after passing through the lymph glands. But the increase of cells is also in inverse ratio with the rapidity of the circulation of the lymph. When this is rapid the cells are hurried on and there is little time for their reproduction. When slow, on the other hand, there is time for cell growth and division in the glandular detention cavities, and the ratio of cells to the plasma is materially increased. Consider next that the multiplicity of cells determines an increase of the fibrine factors, so that the more cells the lymph contains the more material there is for fibrine (Landois), and we have one good reason why under enforced rest the overcharged and congested gland may become the seat of fibrinous coagula or lymphatic embolism. Any over-distension, toxic element or other cause of disturbance, which deranges the functions of the cell, or causes its rapid multiplication by division—as in inflammation—at once sets free the fibrine ferment and determines the coagulation. In the disease before us we have the overfeeding of an animal having a strong digestion, we have an absolute compulsory inactivity, with a sus-

pension to a large extent of the functions of nutrition, sanguification, secretion and elimination; we have in consequence an increase of the blood pressure, and of the solids of the blood and of the plasma of the lymph; we have a suspension of the great motor force of lymph circulation, namely, the muscular contraction, and we have the consequent tardy movement of the lymph, the great increase of lymphocytes, and the distension and engorgement of the lymph glands. As soon as this has reached a certain stage the congestion and incipient inflammation of the gland determines the precipitation of fibrine, the obstruction of the gland, and of the entire circulation of lymph in the lower part of the limb. The fever, the local swelling, and the subsequent steps follow as a matter of course. This view is sustained by the fact that incipient cases can be cured by muscular movement alone. The rarity of the disease in the fore limb may be ascribed to the greater force of the *vis a tergo*, the lesser height of the lymph column, and the stronger action of the aspiratory power of the chest on the lymphatic vessels.

In addition to the causes mentioned above must be noted the following. The disease is an affection of heavy draft horses, in which the tissues are more lax, and the lymph plexus in the connective tissue of the hind limb is much more specious. It is common in the heavy English, Scotch and Belgian draft horses, and rare in the English racer, the American trotter, and in the average light American horse. The malady is more frequent in spring and autumn, when the work is hardest and the feeding most abundant. It rarely attacks the horse in steady work, but appears after an idle Sunday spent in the stable (Monday morning disease), or after one or more days of compulsory idleness from heavy rains or other cause. The damp climate of western Europe has probably an exciting influence, as it has in producing the lymphatic constitution. In the same line of thought Zundel says that many cold weather attacks would be prevented by clipping off the heavy coat which keeps the entire system relaxed. In some cases a sudden change of food, and in others musty oats have been claimed as causes.

Diagnosis. Lymphangitis is distinguished from a simple dropsy of the limb by the acute fever, the great local tenderness especially of the inguinal glands, and by the tender corded

lymphatics that enter these. From cutaneous glanders (farcy) it is diagnosed by the more acute fever, by the swelling of the inguinal glands in the early stage of the disease, followed by the swelling of the lower limb, and by the absence of the hard, comparatively insensible and prone to ulcerate, farcy bud. Farcy buds usually appear on the pastern or fetlock, with more or less swelling of the lower part of the limb, while the inguinal glands are as yet normal in size and without tenderness. From erysipelas, with which this has been confounded, it is distinguished, by the suddenness of the onset, under the circumstances above described, by the high type of fever, by absence of early cutaneous inflammation and the formation of vesicles, and by the fact that lymphangitis commences in swelling of the inguinal glands.

Treatment. In cases that are seen in the earliest stages, before the leg has become badly swollen, recovery will usually take place under active exertion continued for hours at a time. The pumping action inside the hoof during exercise, and the alternate compression and relaxation of the lymph vessels by the muscles, tend to establish a rapid current of lymph, to break up coagula and to re-establish a healthy condition. Friction from below upward on the lymphatic vessels and swollen limb will greatly assist in this restoration. Different agents are employed, such as camphorated spirits in oil, iodine, mercurial, and even blistering ointments. These should not replace exercise when this is possible.

When the fever has set in suddenly and runs very high, the abstraction of four or five quarts of blood, and the administration of a purgative (8 drs. aloes) will be in order. In cases occurring in the same stable and in all other respects apparently identical, the subjects of phlebotomy recovered without any permanent swelling of the limb, while those that were not bled recovered with thickened limb.

In cases so advanced that the limb cannot be used, cold irrigation, with friction, may be applied, and when the irrigation is intermitted one may apply some astringent (vinegar, alum, lead acetate), or an iodine lotion followed by an evenly applied bandage.

The purgative should be followed by full doses of diuretics (nitre, bicarbonate of potash or soda, colchicum, iodide of potassium) until fever and local inflammation have subsided.

As soon as the patient can use the limb, walking exercise should be kept up for several hours forenoon and afternoon.

Throughout the disease the food should be of a light and non-stimulating variety. When appetite returns give at first wheat bran, or roots, or sweet grass in small amount, and do not return to a grain diet until fully recovered and ready to go to work.

After one attack there is always an increased liability to a second, and great care should be taken to give the subject daily exercise, or where this is impossible, to reduce the feed, give a dose of saltpeter, and turn into a yard or roomy loose box on the idle day.

In chronic thickening of the limb, an evenly applied elastic bandage, extending from the hoof up, regular feeding and exercise, washing daily with a weak iodine lotion, and the internal use of iodide of potassium and other diuretics, with bitters and even iron tonics may be used.

INFECTIVE LYMPHANGITIS. TRAUMATIC LYMPHANGITIS.

Infection varied, through wounds, autogenous. Simple irritation, simple lymphangitis. Causes: sun's rays, bruises, other injuries, lymph coagulation from heat, cold, chemical irritants, and coagulants. Germs in blood act on debilitated tissues, lymphatic constitutions, anæmia, overworked or starved, insect bites, claws, teeth of carnivora, foul instruments, fingers or clothes. Bloodless wounds dangerous. Distal parts of the limbs exposed. Fresh wound exposed, granulating less so. Most microbes enter by the lymphatics. Symptoms: extension from wound, swollen lymphatics, reticular lymphangitis, tubular lymphangitis, farcy, tuberculous cases, slough. Fever variable. General infection. Joint infection. Chronic cases. Lesions. Diagnosis, from phlebitis. Treatment: antiseptics, diet, eliminants, antithermics, blisters, mercurial ointment, iodine, lancing, tonics, massage, bandage.

Under this heading must be named not one specific disease but a group of infections entering by the lymphatic vessels and developing inflammation of their substance. They may be divided into two classes, those caused by infection through external

wounds and those in which the poison already in the system becomes localized on a weak or exposed tissue.

A third class must be included, in which there is no recognizable poison but simply a local irritation which leads to coagulation or other alteration in the lymph, or disease of the lymphatic vessels.

The subject belongs rather to surgery than medicine but it seems necessary to contrast it here with the plethoric form of equine lymphangitis. Most of its forms pertain to infectious diseases and will be treated in connection with these.

Causes of Simple Lymphangitis. Formerly many forms of lymphangitis were ascribed to mere local irritation; a superficial form will occur from exposure to the rays of the sun, and an inflammation attendant on a bruise or other injury with unbroken skin, may cause local inflammation of the lymph vessels and enlargement of the adjacent lymph glands. As we have seen above, coagulation of the lymph and fibrine embolism may induce local inflammation in the walls, and this may occur in connection with excessive heat or cold or the presence of chemical irritants and coagulants. These cases are, however, rarely serious and the tendency to-day is to trace nearly all cases to infection, from germs already present in the lymph or blood, or introduced through a wound or sore. The effect of germs already circulating was shown in the beautiful demonstrations of Chauveau in regard to calves subjected to castration by subcutaneous torsion (bistournage). In the healthy calf the simple operation gave rise to little disturbance. The healthy calf injected with septic liquids equally escaped visible trouble. But the calf injected with septic liquids and then subjected to bistournage had a fatal infecting inflammation. There is a strong presumption that, in lymphangitis, starting from an injury with no external sore, the germs were already present in the blood or tissues but were unable to do any serious damage until the injured and weakened part or organ offered an area of lessened resistance to their colonization. Following the same line of thought it has been noticed that animals of a coarse texture, and lymphatic constitution (heavy draft horses and animals raised for the butcher), and such as are debilitated by anæmia, overwork, or poor and insufficient nourishment, are above all liable to be attacked by lymphangitis.

The insertion of the septic poison may take place through the bites of insects, the claws or teeth of carnivora that have been devouring tainted or infecting meat, through the lancet or operating instrument of the surgeon, by his fingers or the dust from his hair or clothes. The wound is perhaps more likely to be infecting if it leads to no effusion of blood, but affects only the thickness of the epidermis, as there is less chance for the washing out of germs by the flowing blood, and there is less care to employ antiseptics. Wounds in the feet and lower parts of the limbs are especially liable to infection by reason of their frequent contact with manure and decomposing organic matter on roads and soil.

A fresh wound, in which the lymph spaces are exposed, is somewhat more open to infection than one that has advanced to the stage of granulation, the layer of unorganized lymph and cells acting as a slight barrier to the passage of microbes.

Nearly all microbial diseases make their inroad by way of the lymphatics, where the sparse cells fail to establish as active phagocytosis as do the numerous moving cells of the blood. Hence a number of infectious maladies are primarily and preëminently diseases of the lymphatics, as glanders, strangles, tuberculosis, cancer, anthrax, swineplague, etc.

Symptoms. The most common form is where lymphangitis extends from some pre-existing wound—as pricked or suppurating foot, fistula of foot, withers or poll, chafing of shoulder or back, cracked heels, boil, sloughing bruise, etc. The swelling around the sore or injury involves in fact the radical lymphatic plexus in the connective tissue (reticular lymphangitis). When the swelling extends and becomes more tense, with firm, painful sinuous cords running out of it in different directions, and especially toward the nearest lymphatic glands, and when these glands are slightly swollen and tender, tubular lymphangitis is diagnosed. No more striking example can be found than in skin glanders (*farcy*). The rigid cords extend from the side of the face, from the eye and nose down toward the submaxillary glands and with more or less adjacent engorgement. Or on a hind limb, or some portion of the trunk, a more or less turgid swelling with one or more firm nodes (*farcy buds*) and painful, tortuous cords running towards the lymph glands is very characteristic.

A tuberculous case may show an indolent, hard, comparatively insensible cutaneous cord leading toward the jugular furrow, the prescapular, precrural or inguinal glands, and at long intervals softening, fluctuating, bursting and discharging a thick pus. In a carcinoma there is the old, hard, nodular, and finally ulcerating swelling from which the firm cords extend to the mass of steadily enlarging lymphatic glands.

A simpler form is where a bruise by the harness causes a hard, thick, slough, embracing the entire thickness of the skin, from which the firm corded lymphatics extend in different directions. After the slow process of detachment, the local lymphangitis usually subsides under simple cooling or antiseptic treatment.

But the grade of such lymphangitis is as varied as the particular germ or combination of germs, present in the wound, and the susceptibility of the animal attacked, and there will be high, moderate or no fever, according to the severity of the case, and in some cases purely local trouble and in others general infection with purulent or septic localization in distant parts. There is always danger of extension to a neighboring joint with destructive results.

A curious outbreak is described by Wiart as attacking nearly every horse in the regiment that sustained a slight wound. A tubercle looking mass formed in the depth of the wound was slow to heal, and the lymphatics leading out from it became round, corded, turgid, and at long intervals developed along their course fluctuating centres which, whether opened spontaneously or by the lancet, showed the same indolent habit. A single attack would last from two to six months, and the actual cautery had to be used on the sores.

The *lesions* are those already described in the last article for simple lymphangitis. For infecting cases they are those of the particular disease which may be present.

Diagnosis. The general diagnosis of lymphangitis is the distinction from phlebitis. In phlebitis the vein is blocked and can not be raised by pressure on the side leading toward the heart; in lymphangitis it can be so raised. The swelling and tenderness are both greater in lymphangitis. The inflamed vein is more rectilinear, the lymph vessel somewhat sinuous. If suppuration ensues it is more diffuse in lymphangitis; more restricted and mixed with the elements of blood in phlebitis.

For identification of the particular forms of infecting lymphangitis, the reference must be made to the individual infectious diseases.

Treatment. In general the treatment of lymphangitis is the antiseptics of wounds. Further than this the treatment of each case is that of the particular disease which it represents. For all cases alike it is important to apply vigorous treatment early, so as to cut it short before it can attain a dangerous extension.

For the simpler forms of lymphangitis the wound should first be thoroughly cleaned and disinfected. Washing with soap suds, or solution of carbonate of soda will remove any greasy agent which would prevent a thorough antiseptics. Then it may be washed with the antiseptic lotion :—carbolic acid solution (1:20), or mercuric chloride solution (1:500), or zinc chloride (1:400), or potassium permanganate (1:160), or bismuth subcarbonate (pure) may be applied in glycerine. If the infection has been introduced by a small or punctured wound, the sting or bite of an insect, or the prick of a sharp instrument it should be freely cauterized to its depth with lunar caustic incising it if need be to reach the whole of the poison, and the surface afterward dressed with antiseptics.

The diet should be light but nutritious and laxative, and the free action of the bowels and kidneys should be maintained by salines. When fever runs high give quinine, or salicylate of soda. When a large wound has to be dressed it may be requisite to use a non-poisonous agent like acetate of aluminium or boric acid to irrigate it thoroughly. In some such cases packing the irrigated wound with iodoform gauze or aristol has an excellent effect.

When there is a firm inflamed cord, hot and painful, a fly blister along its course followed by mercurial ointment often gives excellent results. Or the cord may be repeatedly painted with solution of silver nitrate or tincture of iodine.

Foci of suppuration must be promptly opened and thoroughly and persistently disinfected.

With suppuration in multiple abscesses or large open sores liberal feeding must be enjoined and iron and other tonics should be resorted to.

The persistent swelling of the part must be met by active rubbing or kneading, by exercise and by uniform compression by a flannel or elastic bandage.

SACCHAROMYCOSIS FARCINOIDES. MYCOTIC LYMPHANGITIS.

See Vol. IV.

LYMPHANGIECTASIS. DILATED LYMPHATICS.

Result of lymphangitis, of heart disease, of pulmonary arterial thrombosis, of external jugular plugging. Causes : obstruction to lymph flow, compression, increased venous blood pressure, fibrinous lymph coagula, action of sensory nerves, of lymphadenitis, anæmia. Symptoms : like dropsy if in plexus, in large lymphatics, moniliform swelling, sacculation, wounds discharge lymph, hyperplasia of connective tissue, fatty deposits, lipomata. Treatment : elastic bandage, cold astringents, iodine, punctures, ligatures, cauterizations, tonics.

The most striking cases of dilatation of the lymphatics in the lower animals are met with in horses that have suffered repeatedly and severely from the lymphangitis of plethora. Then the lower part of the shank and the pastern are enormously thickened to perhaps two or even three feet in circumference, and skin and connective tissue are the seat of a general dilatation of the lymphatic plexus and vessels with great thickening of their walls. Nocard and Barrier record cases of general dilatation of the lymphatics in dogs, in connection with heart disease, also the case of a horse with old standing thrombosis of the pulmonary arteries, hypertrophy of the right heart, and dilatation of the thoracic duct to the size of the arm, and of the lymphatics of the mesocolon to the diameter of half an inch to nearly an inch. Nocard records two cases in the horse, one of a reticular lymphangioma of the sheath, and the other of dilatation of the lymph vessels accompanying the saphena vein on the inside of the thigh. This formed small, soft, fluctuating, extremely irregular tumors, completely covering the vein for a space of about four inches.

In both cases the dilatations were surrounded by a thick layer of connective tissue filled with liquid. Virchow records a case of a new-born calf in which a thrombosis of the external jugular vein caused obstruction of the mouth of the thoracic duct, and a consequent extreme distension of all the splanchnic lymph vessels with a slightly sanguinolent fluid. The intestines especially

were covered everywhere with broad, bead-like canals, arranged so closely together that the intervening tissue could scarcely be recognized.

The *causes* of lymphangiectasis appear to be generally some obstruction to the onward flow of the lymph. Any diseased condition, therefore, that causes compression of the larger lymph vessels may cause dilatation of the smaller ones leading into these. General distension may come from disease of the lungs or left heart and increased venous blood pressure, or from thrombus of the jugular, or a tumor obstructing the thoracic duct, while local engorgements may come from the pressure of tumors, or the occurrence of lymphangitis and formation of fibrinous coagula. In cases of partial obstruction of the lymph vessels, the increased secretion of lymph may lead to distention and enlargement. It may be named in this connection that irritation of the sensory nerves in dogs has been shown to determine a larger production of lymph (Krause). Lymphadenitis and the obstruction of the passage of lymph through the glands is an obvious cause, and hence the disease is specially liable to appear in connection with diseases which show a predilection for the lymphatics (tuberculosis, glanders, strangles, carcinoma, etc).

In his work on dilatation and occlusion of lymph channels, Busey shows that in man the majority of cases are in hospital patients in whom blood and general health have been impoverished and reduced by unhygienic conditions. One case gave support to the theory of maternal impression, the pregnant mother having suffered from over-use of the right limb on a sewing machine, and the offspring having shown extensive lymphangiectasis in the right leg.

Symptoms consist in enlargement of the lymph vessels or plexus, and often of the glands. If of the lymph plexus, it may appear like a dropsical effusion in the part, with or without sacular dilatations at intervals. If of the larger vessels, their tortuous anastomosing trunks, following largely the lines of the veins, are usually characteristic. If the distention is slight it is usually moniliform, as the valves are still intact, and the intervals between stand out as bladder-like masses. If the structure is wounded or if it ulcerates there is the discharge of a straw-colored fluid, often rendered milky by the presence of fatty gran-

ules, and at times tinged with blood. There is always a tendency to the increase and condensation of the connective tissue surrounding the vessels, and fatty degeneration and the formation of lipomata are not uncommon,

Treatment. Compression, by flannel or elastic bandage, from the foot upward, is the simplest and most promising treatment when the limb is affected. The local application of cold astringents or iodine may be added. Punctures, ligatures, and cauterization have not given encouraging results. Ligature of the nutrient artery of the part, has succeeded in one or two cases, but has failed in others. Tonics are to be tried more particularly in cases due to specific debilitating diseases. Sometimes a spontaneous recovery has been noticed when the surrounding connective tissue has increased and contracted in connection with inflammation.

LYMPHORRHŒA. LYMPHORRHAGIA. DISCHARGE OF LYMPH THROUGH WOUNDS OR SORES.

Result of rupture of lymphatics. Milky, fatty lymph. Treatment : ligature, excision, cauterization, of little avail, compression, tonics.

Obstruction of a lymph duct may lead to rupture and the discharge of its fluid on the surface or into an internal cavity. Dr. Cayley records a case of fatal peritonitis in man from rupture of the receptaculum chyli, and the formation of lymph fistulæ has been attributed to *filaria sanguinis hominis*. We are aware of no corresponding case in connection with the blood parasites of the horse or dog. In the larger domestic animals the great thickness and resistance of the skin offers a barrier to the rupture of subcutaneous lymph vessels, but this no longer applies in case of a suppurating or ulcerous wound. The escaping lymph has often a milky hue from the admixture of fat, just as its escape in the kidneys causes chyluria, and in the bowel fatty stools. The escape is often very profuse and persistent, and results in marked debility. Ligature and excision of the fistulous vessel, also caustics—actual and potential, have been tried with rather poor success. Fitzer succeeded in an obstinate case by the extensive application of nitrate of silver and others by simple compression. As the victims are mostly debilitated a course of tonics is usually desirable.

LYMPHADENITIS. INFLAMMATION OF THE LYMPH GLANDS.

Result of lesions of tributary tissues. Arrest in glands of microbes and other irritants. Trauma of gland. Inflammation. Symptoms: swelling, stiffness, gland tender, hot, pitting envelope, corded lymph vessels, abscess, fever. Lesions. Treatment: antiseptics, astringents, emollients, vesicants, lancing, antiseptics, antiphlogistics, antithermics. Chronic adenitis. Symptoms: enlarged glands without engorgement, if simple affects a single gland, if infectious, a group. Lesions: gland swelling, induration, shrinking, follicular distension, pigmentation, growth of lymphocytes, caseation, calcification. Treatment: antiphlogistic, antiseptic, iodine, chloride of calcium, iodide of potassium.

Apart from traumatic lesions, lymphadenitis virtually implies some lesion of the tissues from which the different vessels of the glands proceed. The glands, however, have been referred to as filtering agents on the course of the lymph vessels and in this partial view of their functions we find abundant reason why irritants, carried in the lymph stream, should be arrested with pathogenic results in the glands. A particle of pigment, gaining entrance to the lymph vessels, tends to be arrested among the trabeculæ of the gland, and contributes to the pigmentation so common in old animals. Cells and granules from malignant tumors, and bacteria from an infection-atrium are arrested in the glands and make these the great centres of infection lesions.

Traumatic inflammation comes from bruises, lacerations, punctures or incisions directly implicating the glands. There result swelling, tenderness and the other general signs of inflammation, and in the case of an open wound, possibly lymphorrhagia.

Acute inflammation more commonly supervenes on inflammation in the area drawn upon by the afferent vessels of the gland. In inflammations generally, the adjacent lymphatic glands become congested. In lymphangitis it is so in a marked degree. In external parts we can follow this by careful observations during life, in internal organs we often find the glandular enlargement after death.

Symptoms consist in swelling and perhaps stiffness in the region of the gland. Manipulation shows tenderness and heat, the gland being felt abnormally large, round, or oval, tense, loose

from the skin but having a distinct envelope of soft pitting exudate which tends to increase in a downward direction. There may or may not be a corded feeling of the afferent lymphatic trunks. As the pasty swelling increases, it extends into surrounding parts, binds the gland to the skin and adjacent structures, and may even conceal the gland in the excess of its investing engorgement. This is especially frequent in strangles. As the process advances softening may take place in the centre and extend toward the circumference, and this may burst like an ordinary abscess. In some cases the softening is very limited and tardy, and the pus may be pent up and inspissated, or it may appear to be entirely reabsorbed, while the gland is in process of induration. Fever, which may run high during the process of suppuration, moderates when that has been accomplished.

In the case of glands too deeply seated to be clearly felt, the occurrence of purulent fluctuation in their vicinity suggests abscess of the glands, an important induction, as the maturation and healing are usually slow in the gland tissue.

Lesions. At the outset the glands are visibly enlarged, softened, and of a dark red hue, with spots of a brighter red. The changes, mainly in the medullary layer, consist in a great proliferation of spheroidal cells in the follicles and also of polyhedral cells in the lymph sinuses. The endothelial cells are swollen, the blood vessels gorged, and extravasations of blood into the follicles and sinuses are frequent. Abscess or fibroid hyperplasia with induration may follow. Much depends on the particular infection (tuberculosis, glanders, carcinoma, etc.) as the special product of each disease will be found in the affected gland.

Treatment is in the main as advised for lymphangitis and will vary with each specific causative disease. Locally, antiseptics, astringents, deobstruents, emollients, and vesicants, will be requisite in different cases. As soon as pus can be distinctly diagnosed it should as a rule be evacuated, and the cavity treated antiseptically. General treatment may at first be antiphlogistic and febrifuge, but must usually embrace tonics and stimulants in the end.

Chronic Adenitis may be a sequel of the acute, or it may arise independently. In the latter case it is usually the result of some other disease (tuberculosis, glanders, carcinoma, sarcoma,

melanosis, inveterate disease of the skin, chronic fistula, abscess, or mucous inflammation).

The *symptoms* are those of enlarged glands with no material surrounding engorgement. In the infections of tuberculosis and glanders it shows a tendency to affect the whole group, whereas in simple abscess or in suppuration of the nasal sinuses it may implicate one gland only, the remainder appearing normal.

Lesions. The gland often becomes indurated and even shrunk, the connective tissue elements undergoing a steady increase at the expense of the follicles and lymphoid cells. This is a common condition of tuberculous glands (perl-knoten, grapes) of cattle, but may result from the entrance of pigment or other cause of mild irritation. In other cases, pigment entering from without or developed from blood in the congested gland, finds permanent lodgment in its tissue and may give it a gray mottled or quite black aspect. In still other cases, there is a great increase of the round lymphoid and larger polyhedral cells, many of which degenerate, becoming strongly refracting, stain feebly, or not at all, and pass into a cheesy degeneration. This is a common condition in tuberculosis and glanders, and the caseous centres, beginning as multiple miliary centres, many coalesce to form masses of six or twelve inches in their greatest diameter, as in bovine tuberculosis. In other cases the caseating mass becomes the seat of calcareous deposit and the necrotic and caseated gland becomes in part calcified. Other degenerative changes such as atrophy, amyloid, and hyaline are met with but have received little attention.

Treatment will be subordinated to the primary cause. If that is a simple local inflammation or irritation its removal will entail a speedy improvement in the gland, and, in the absence of too extensive structural change, a rapid recovery. The infectious cases, on the other hand, are likely to prove as inveterate as the disease on which they depend. In case the enlargement or congestion of the gland persists after the removal of its primary cause, local deobstruents, especially the preparations of iodine, are usually effective. Tincture of iodine with soap, iodide of lead, and mercurial ointment have been severally used with advantage. Injection of a weak solution of iodine into the gland will at times succeed. The internal use of chloride of calcium or iodide of potassium will often hasten recovery.

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